The Slt2/Mpk1 mitogen-activated protein kinase (MAPK) cell integrity pathway is involved in maintenance of cell shape and integrity during vegetative growth and mating in *Saccharomyces cerevisiae*. Slt2 is activated by dual phosphorylation of a threonine and tyrosine residue in response to several environmental stresses that perturb cell integrity. Negative regulation of Slt2 is achieved via dephosphorylation by two protein-tyrosine phosphatases, Ptp2 and Ptp3, and a dual specificity phosphatase, Msg5. In this study, we provide genetic and biochemical evidence that the stress-inducible dual specificity phosphatase, Sdp1, negatively regulates Slt2 by direct dephosphorylation. Deletion of *SDP1* exacerbated growth defects due to overexpression of Mkk1<sup>ΔS464</sup>, a constitutively active mutant of Slt2 MAPK kinase, whereas overexpression of Sdp1 suppressed lethality caused by Mkk1<sup>ΔS464</sup> overexpression. The heat shock-induced phosphorylation level of Slt2 was elevated in an *sdp1Δ* strain compared with that of the wild type, and heat shock-activated phospho-Slt2 was dephosphorylated by recombinant Sdp1 in vitro. Under normal growth conditions, an Sdp1-GFP fusion protein was localized to both the nucleus and cytoplasm. However, the Sdp1-GFP protein translocated to punctate spots throughout the cell after heat shock. *SDP1* transcription was induced by several stress conditions in an *Msn2/4* dependent manner but independent of the Rlm1 transcription factor, a downstream target activated by Slt2. Induction of *SLT2* by high osmolality was dependent on Rlm1 transcription factor and Hog1 kinase, suggesting cross-talk between Slt2 and Hog1 MAPK pathways. These studies demonstrate regulation of Slt2 activity and gene expression in coordination with other stress signaling pathways.

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Mitogen-activated protein kinase (MAPK) pathways are evolutionarily conserved signal transduction cascades connecting extracellular stimuli to a wide range of cellular responses. The MAPK cascades are sequential phosphorylation-mediated activation of three kinases, MAPK kinase kinase, MAPK kinase, and MAPK (1, 2). Activation of MAPK requires phosphorylation of both threonine and tyrosine residues of a TXY motif in the activation loop. Therefore, inactivation of MAPK can be achieved by dephosphorylation of either of the two phosphorylation sites. It has been demonstrated that three types of phosphatases, protein-tyrosine phosphatase, serine/threonine phosphatase, and dual specificity phosphatase, are involved in negative regulation of MAPK from yeast to mammals (3).

*Saccharomyces cerevisiae* encodes five MAPKs involved in distinct cellular responses (4). The four MAPKs present in vegetative cells, Fus3, Kss1, Hog1, and Slt2/Mpk1, are involved in the mating-pheromone response, filamentation-invasion pathway, high osmolarity growth, and cell integrity pathway, respectively. It has been known that two protein-tyrosine phosphatases, Ptp2 and Ptp3, a dual specificity phosphatase Msg5, and type 2C serine/threonine phosphatases are involved in differential inactivation of distinct MAPKs. Ptp3 dephosphorylates Fus3 to maintain a low basal activity and to inactivate Fus3 following pheromone stimulation, and its homologue Ptp2 also plays a minor role as a Fus3 phosphatase (5). Ptp2 and Ptp3 are also involved in negative regulation of Hog1 and Slt2 for maintaining low basal activities and for adaptation following osmotic stress and heat shock, respectively (6–9). However, for Hog1 and Slt2 kinases, Ptp2 is more effective than Ptp3. Targets of the Msg5 dual specificity phosphatase include Fus3, Slt2, and Kss1 (5, 10–12). Msg5 is involved in recovery from pheromone-induced G<sub>1</sub> arrest by dephosphorylating Fus3 kinase (11). The type 2C protein phosphatase Ptc1 inactivates Hog1 for maintaining low basal levels of Hog1 activity and adaptation in response to osmotic stress (13). A genetic interaction suggests an involvement of Ptc1 in the protein kinase C pathway; however, there is currently no evidence for direct dephosphorylation of Slt2 by Ptc1 (14).

The Slt2 cell integrity pathway is involved in maintenance of cell shape and integrity during vegetative growth and mating. This pathway is activated by several environmental stimuli such as heat shock (15), hypoosmotic stress (16), mating pheromone (17), agents causing cell wall stress (18), and actin remodeling (19). Putative sensors of the Slt2 pathway are the transmembrane proteins Wsc1 (20) and Mid2 (21), which interact with the GDP/GTP exchange factor Rom2 to stimulate GTP loading of the small GTP-binding protein Rho1 (22). Rho1 binds and activates Pck1, which elicits serial activation of the Slt2 MAPK module composed of MAPK kinase kinase (Bck1), two redundant MAPK kinases (Mkk1/Mkk2), and a MAPK (Slt2) (23). The Rlm1 (24) and SBF (25, 26) transcription factors are two downstream targets of Slt2. Most Rlm1-regulated genes encode cell wall proteins or enzymes involved in cell wall biosynthesis (27). SBF is a heterodimer complex composed of the Swi4 and Swi6 proteins, which regulates gene expression during the G<sub>1</sub>/S transition of the cell cycle (28). SBF-activated genes are involved in budding and in membrane and cell wall biosynthesis (29). It has been shown that Slt2 is down-regu...
lated by the phosphatases Ptp2, Ptp3, and Msg5. However, little is known about the precise mechanism of negative regulation of Slt2.

Since the completion of the genome sequence of *S. cerevisiae*, genomic scale analyses have provided a wealth of information to experimentally examine the potential function of newly identified proteins and their interactions. One of the reports analyzing global protein-protein interactions using two-hybrid screens suggested an interaction between the Slt2 kinase and the protein Yil113w (renamed as Sdp1), a potential dual specificity phosphatase (30). In addition, Yil113w, a putative dual specificity phosphatase (30), was found by a standard protocol. GST-Sdp1 fusion protein was cleaved with Factor Xa protease, and the GST protein was removed by incubation with glutathione-agarose (Sigma). The presence of Sdp1 in yeast cells was confirmed by immunoprecipitation with anti-HA antibody to determine the levels of Slt2–3HA.

**Co-immunoprecipitation—**Co-immunoprecipitation of Sdp1–13MyC and Slt2–3HA was carried out as follows. JH21 strain, in which the SDP1 gene was replaced by SDP1–13MyC, was transformed with Yep352–SLT2–3HA or Yep352 plasmid. Cells were grown in SC-Ura medium up to A600 of 0.7 at 25 °C and then shifted to 39 °C for 1 h. Both heat-pretreated and untreated cells were broken in immunoprecipitation buffer (50 mM HEPES (pH 7.6), 100 mM NaCl, 0.5% Nonidet P-40) with protease inhibitors. 1 mg of cell extract was immunoprecipitated with anti-HA antibody and protein A-Sepharose. The precipitates were washed five times with an immunoprecipitation buffer, resuspended in 50 μl of 2× SDS sample buffer, and boiled for 5 min. The electrophoresis was performed on an immuno-blot analysis with anti-HA antibody or anti-Myc (9E10; Roche Molecular Biochemicals) antibody.

**Northern Blot Analysis—** Yeast cells were grown in YPD medium to A600 of 0.7 at 30 °C and shifted to 39 °C for heat shock. For oxidative stress or osmotic stress, a final concentration of 0.3 mM H2O2 or an equal volume of YPD containing 2 mM sorbitol was added to the culture and incubated further at 30 °C. PCR-amplified open reading frames of SLT2, MSG5, SDP1, and ACT1 were used as probes for Northern blotting. For the detection of PTP2 and PTP4 mRNA, 0.7-kb SpeI/NotI and 0.9-kb XbaI/HindIII internal fragments were used, respectively.

**Fluorescence Microscopy—**To localize Sdp1 and Slt2, BY4743 diploid sdp1Δ cells containing pRS425–SDP1–GFP and slt2Δ cells containing pRS425–SLT2–GFP were grown in SC-Leu medium. 10 μg/ml 4,6-diamidino-2-phenylindole was added for nuclear staining, and the fluorescence images were detected using a Nikon Eclipse E800 fluorescence microscope equipped with a Hamamatsu ORCA-2 cooled CCD camera.

**RESULTS**

**Sdp1 Phosphatase Regulates the Slt2 Cell Integrity Pathway—**To test the possible role of the Sdp1 (Yil113w) dual specificity phosphatase in regulation of Slt2 kinase, we examined whether deletion or overexpression of SDP1 could affect growth defects caused by hyperactivation of the Slt2 pathway. Overexpression of the Mkt1pp36 allele, which was originally identified as a suppressor of the cell cycle phenotype of a bck1Δ strain, inhibits growth by constitutive activation of the downstream MAPK Slt2 (24, 34). *S. cerevisiae* strains were transformed with plasmid pNV7–MKK1pp36 (P*GAL1*, MKK1pp36) (24), in which the MKK1pp36 allele is under the control of the GAL1 promoter, and tested for growth on medium containing glucose (repressing) or galactose (inducing) (Fig. 1A). A growth defect observed on galactose medium by MKK1pp36 overexpression was exacerbated in ptp2Δ and msg5Δ deletion strains compared with the wild type as previously reported (8, 24). Although a previous report indicated no difference in growth between wild type and ptp3Δ strains upon MKK1pp36 overexpression (8), we could observe a modest growth defect in the ptp3Δ strain under our experimental conditions, perhaps reflecting a minor contribution of Ptp3 in Slt2 phosphorylation compared with Ptp2. The effect of SDP1 deletion on the MKK1pp36 phenotype was weaker than that of the PTP3 deletion, but the sdp1Δ strain also showed poorer growth than the wild type on galactose medium with virtually indistinguishable growth on glucose medium.

As a corollary experiment, we tested whether overexpression of Sdp1 could suppress the slow growth phenotype of yeast cells expressing Mkk1pp36. Expression of SDP1 both on a low copy number plasmid (pRS415) and a high copy number plasmid
Following hyperactivation of the Slt2 pathway and Slt2 is activated by phosphorylation, we investigated the possibility that phosphatase substrate p-nitrophenyl phosphate (data not shown). Furthermore, Sdp1 dephosphorylated Slt2–3HA that had been immunoprecipitated from heat-shocked yeast cells (Fig. 3). Incubation with sodium orthovanadate, an inhibitor of protein-tyrosine phosphatases, inhibited dephosphorylation of Slt2–3HA by Sdp1 in vitro. Taken together, these results suggest that Sdp1 functions in the Slt2 pathway by direct dephosphorylation of Slt2 kinase.

**Sdp1 Interacts with Slt2 in Vivo—**Data described above suggest a functional interaction between the Sdp1 phosphatase and the Slt2 kinase. Although genome-wide two-hybrid analysis suggested an interaction between a Gal4 activation domain (GAD) fusion of Slt2 and Gal4 DNA binding domain (GBD) fusion of Sdp1 (30), it was necessary to confirm their interaction to rule out the possibility ofa false positive interaction. *S. cerevisiae* SFY526 strain containing an integrated GAL1-lacZ reporter was transformed with plasmids expressing GBD-Sdp1 and GAD-Slt2 or GBD-Sdp1 and VP16-Slt2 fusion proteins, and the interactions were detected by β-galactosidase assays (Fig. 4A). Although the overall activity was low, transformants expressing GBD-Sdp1 and GAD-Slt2 (1.3 Miller units) or GBD-Sdp1 and VP16-Slt2 (2.3 Miller units) showed higher activity than the cells containing GBD-Sdp1 alone (0.1 Miller units) or GAD-Slt2 alone (0.2 Miller units), suggesting an interaction between Slt2 and Sdp1.

We also tested the interaction between Sdp1 and Slt2 by co-immunoprecipitation experiments from yeast cell extracts. For this purpose, we constructed a genomic tagged version of Sdp1 encoding 13Myc epitopes at the carboxyl terminus. The 13Myc-tagged Sdp1 was fully functional as ascertained by the resistance of cells expressing Sdp1–13Myc to toxicity due to Mkk1p386 overexpression (data not shown). Cells expressing Sdp1–13Myc were transformed with YEp352-SLT2–3HA or vector control, and the transformants were grown at 25 °C and heat-shocked at 39 °C for 1 h before harvest and protein extraction under native conditions. Slt2–3HA was immunoprecipitated with anti-HA antibody and immunoblotted with anti-Myc antibody. This experiment revealed co-immunoprecipitation of Sdp1–13Myc with Slt2–3HA (Fig. 4B). The
strength of the interaction, at least by this analysis, was not responsive to heat shock.

The Localization of Sdp1 Changes upon Heat Shock—To localize Sdp1 within yeast cells, we expressed a functional Sdp1-GFP fusion protein in a diploid sdp1Δ strain. At 25 °C, Sdp1-GFP was localized in both the nucleus and cytoplasm with slight accumulation in the nucleus (Fig. 5A). Upon heat shock, the Sdp1-GFP fusion was observed in punctate spots throughout the cells. This relocalization of Sdp1-GFP was observed within 5 min after heat shock, and after 30 min of heat shock, the localization pattern was more intense and punctate. The punctate Sdp1-GFP spots were redistributed evenly throughout the cells when the heat-shocked cells were shifted back to 25 °C for 20 min. This translocation of Sdp1 was quite specific to heat shock and Sdp1-GFP, since we could not detect changes in Sdp1-GFP localization after treatment with 0.3 mM H₂O₂ or 1 M sorbitol (data not shown). We also localized Slt2 using a functional Slt2-GFP fusion, which can complement the temperature-sensitive phenotype of an slt2Δ mutant. In accordance with the previous report localizing Slt2-HA (35), Slt2-GFP was concentrated in the nucleus and also located in the cytoplasm at 25 °C (Fig. 5B). Although a previous report showed more uniform distribution of Slt2-HA between the cytoplasm and the nucleus after heat shock (35), the Slt2-GFP fusion showed little change in localization 30 min after heat shock.

Differential Expression of SLT2 and Phosphatase Genes in Response to Stress Conditions—Slt2 is regulated by at least four protein phosphatases, Ptp2, Ptp3, Msg5, and Sdp1. These phosphatases show diverse specificity toward other MAPKs. Ptp2 and Ptp3 are also involved in dephosphorylation of Fus3 and Hog1, and Msg5 also dephosphorylates Fus3 and Kss1 (6, 7, 30). In addition to specific interactions between the phosphatases and kinases, the abundance of individual phosphatases is likely to be an important factor determining the specificity of the phosphatases under distinct stress conditions that activate specific MAPKs.

Since the SDP1 and PTP2 promoters contain stress response elements, which are the binding sites for the general stress transcription factors Msn2/4 (36), we investigated whether Msn2/4 are involved in the expression of SLT2 and phosphatase genes under a number of stress conditions. We compared stress-inducible gene expression patterns between the W303–1A wild type strain and an isogenic msn2msn4 mutant (Fig. 6A). As previously established, SLT2 and PTP2 steady state mRNA levels were induced by heat shock (8, 27). These two genes showed slow induction kinetics compared with SDP1 induction, which peaked at 10 min after heat shock. The SLT2, PTP2, and SDP1 genes were also induced by H₂O₂ treatment. In contrast, expression of PTP3 and MSG5 was transiently decreased upon heat shock, and there was little change in expression by H₂O₂ treatment. The SLT2 and all the phos-
Phosphatase genes tested (PTP2, PTP3, MSG5, and SPP1) were induced by osmotic stress 30 min after treatment. Only SDP1 was dependent on Msn2/4 for induction by heat, H2O2, and osmotic stress via the administration of 1M sorbitol. These data suggest differential regulation of SLT2 and phosphatase genes under different stress conditions.

Previous studies have shown that the Rlm1 transcription factor, which is activated by Slt2-dependent phosphorylation, is responsible for SLT2 mRNA induction in response to heat shock (24, 27, 37). In addition, it has been shown that induction of PTP2 by heat shock is dependent on Slt2 (8). To examine the contribution of Rlm1 in the activation of SLT2 and phosphatase genes in response to oxidative stress, osmotic stress, and heat shock, we compared stress-inducible expression of these genes in isogenic wild type (BY4741), slt2Δ, and hog1Δ strains. PTP2 induction was largely, although not completely, dependent on Rlm1. Expression of PTP3 and SDP1 in response to stresses was not significantly changed in the rlm1Δ strain. The residual heat shock induction of SDP1 observed in msn2Δmsn4 (Fig. 6A) was still detected in an msn2Δ msn4 rlm1Δ mutant strain (data not shown). Therefore, the stress-inducible expression of SDP1 is independent of the Rlm1 transcription factor but partially dependent on Msn2/4-mediated activation in response to heat shock, H2O2, and osmotic stress.

It has been previously demonstrated that Slt2 and Hog1 respond in opposite ways to osmotic changes. Slt2 is transiently activated in response to hypoosmolarity, whereas Hog1 is activated by hyperosmolarity (16). Since SLT2 mRNA was induced by 1M sorbitol in an Rlm1-dependent manner, we hypothesized that there might be an upstream regulator other than Slt2 to activate Rlm1 in response to hyperosmotic stress. Mlp1 was identified as an Rlm1-interacting protein that has...
mRNA induction by 1 M sorbitol was significantly reduced in a hog1 mutant strain (data not shown). In a previous report, induction of Rlm1 on heat shock-induction of PTP2 suggests cross-talk between the Slt2 and cAMP-PKA pathways, whereas Hog1-dependent induction of Slt2 under hyperosmotic conditions might establish linkage between the Slt2 and Hog1 MAPK pathways in transcription.

**DISCUSSION**

The Sdp1 Dual Specificity Phosphatase Inactivates Slt2 by Direct Dephosphorylation—The Slt2 MAPK pathway is essential to maintain cell wall structure during vegetative growth and mating and in response to environmental stresses that perturb cell wall integrity. Therefore, Slt2 activity must be orchestrated with cellular processes such as the cell cycle, pheromone responses, and stress responses.

Including the Sdp1 dual specificity phosphatase, which was demonstrated to negatively regulate Slt2 in the present study, four phosphatases have been identified as regulators of Slt2. Two protein-tyrosine phosphatases, Ptp2 and Ptp3, and two dual specificity phosphatases, Msg5 and Sdp1, appear to have redundant roles as well as specific roles in the regulation of Slt2. All of the phosphatase deletion strains tested, especially ptp2Δ/ptp3Δ (8) and msg5Δ (10), showed higher basal phosphorylation levels of Slt2 than the wild type, suggesting a role for these phosphatases in regulating basal activity of Slt2. In addition, heat-induced Slt2 phosphorylation was also increased in ptp2Δ, sdp1Δ (Fig. 2), ptp2Δ/ptp3Δ (8), and msg5Δ (10) strains, implying the potential involvement of these phosphatases in the down-regulation of Slt2 activity for adaptation after stress. Since Slt2 can be activated by various stress stimuli other than heat shock, the role of the four phosphatases might be differentiated under specific stress conditions. It remains to dissect out specific roles of these phosphatases in regulation of Slt2 and other target kinases.

It has been shown that protein-protein interaction through amino-terminal noncatalytic domains of Ptp2 and Ptp3 determines their substrate specificity toward Hog1 and Fus3 (40). In addition, the localization of phosphatases could affect their specificity toward various MAPKs. Ptp2 is nuclear, whereas Ptp3 is cytoplasmic and excluded from the nucleus (8). Moreover, the Ptp2 and Ptp3 can regulate Hog1 localization by tethering Hog1 in the nucleus and cytoplasm, respectively (9). At 25 °C, Sdp1-GFP was observed throughout the cell with slightly enhanced nuclear localization. Interestingly, Sdp1-GFP showed rapid translocation to punctate spots after heat shock, implying association of Sdp1 with subcellular organelles or with other proteins. Identification of Sdp1 substrates other than Slt2 will help to identify the location of Sdp1 after heat shock, and the significance of the translocation event.

**Expression of PTP2 and SDP1 mRNA upon Heat Shock**

Forms Feedback Regulation of the Slt2 Pathway and Cross-talk between Slt2 and cAMP-PKA Pathways—The expression of many phosphatases that act upon MAPKs is under the control of their target MAPKs or upstream signals activating MAPKs, forming feedback regulation loops (3). In accordance with the previous report showing Slt2-dependent induction of PTP2 by heat shock (8), we showed that Slt2 and its downstream transcription factor Rlm1 is largely involved in heat shock induction of PTP2. SDP1 was induced by various stress conditions such as heat shock, oxidative stress, and osmotic stress in an...
Msn2/4-dependent manner but independent of the Slt2 pathway. Msn2/4 transcription factors regulate genes containing stress response elements, and their activity is negatively regulated by the cAMP-PKA pathway, which is involved in nutrient signaling (41). The putative sensors of the Slt2 pathway, Wsc1 and Wsc2, were isolated as multicopy suppressors of heat shock sensitivity of ira1Δ strain in which hyperactivation of Ras causes an increase in cAMP production, suggesting possible cross-talk between the Slt2 and the cAMP-PKA pathways (20). Taken together, under heat shock conditions, Rlm1-dependent induction of PTP2 forms feedback regulation of Slt2 activity, whereas regulation of Slt2 activity by Sdp1 whose expression is dependent on Msn2/4 would provide linkage between the Slt2 and cAMP-PKA pathways (Fig. 7A).

Hog1 Regulates Rlm1-dependent SLT2 Expression upon Osmotic Stress—It has been known that Rlm1, whose activity is regulated by Slt2, mediates heat shock induction of Slt2-regulated genes including SLT2 itself (27). Most of the Rlm1-regulated genes identified by genome-wide analysis encode cell wall proteins or enzymes involved in cell wall biosynthesis (27). Furthermore, although it remains to be confirmed, putative Rlm1-binding consensus elements are identified on the promoters of genes involved in Slt2 pathway such as MID2, SAC7, BCK1, M KK1, and RLM1 itself. We demonstrated here that Rlm1 is responsible for not only heat shock induction but also H$_2$O$_2$ and osmotic stress induction of SLT2. We also showed that Hog1 kinase is involved in induction of SLT2 by hyperosmotic stress, suggesting cross-talk between Hog1 and Slt2 kinase pathways. Hog1 and Slt2 kinases are activated by hyperosmolarity and hypoosmolarity, respectively. Although Hog1 and Slt2 kinases are regulated in opposite directions by changes in external osmolarity, it is likely that they are coordinately regulated. Dephosphorylation of Hog1 by hypoosmotic stress has been shown to be dependent on Slt2 pathway (16). The biological significance of Hog1-dependent induction of SLT2 is not yet clear; however, it might reflect requirements for cell wall changes after adaptation to hyperosmotic stress. Induction of some cell surface proteins or cell wall biosynthetic enzymes by hyperosmotic stress supports this idea (38).

Hyperosmotic stress induction of SLT2 is dependent on Hog1 kinase and the Rlm1 transcription factor. These data suggest that the Hog1 kinase pathway may regulate Rlm1 activity under high osmolarity conditions (Fig. 7B). Rlm1 is likely to be partially involved in osmotic induction of PTP2 to form feedback regulation of Hog1. Osmotic stress induction of PTP3 might be mediated by yet unidentified transcription factors that are regulated by Hog1. Rlm1 is a member of the MADS (Mcm1, Agamous, Deficiens, SRF) box family of transcription factors, which have a conserved amino-terminal MADS box DNA binding domain (24). One of the characteristics of MADS box proteins is their interaction with co-regulators to regulate gene expression. Two-hybrid screening identified Mip1, a homo-logous protein to Sit2, as an Rlm1-interacting protein (24). Although there is no evidence that Rlm1 interacts with other proteins than Sit2 and Mip1, it is still possible that additional accessory proteins are involved in regulation of Rlm1 activity under different conditions. Further studies are necessary to determine whether Hog1 could regulate Rlm1 directly or indirectly by regulating interacting partners and how Rlm1 is differentially regulated under heat shock and osmotic stress conditions.

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Regulation of the *Saccharomyces cerevisiae* Slt2 Kinase Pathway by the Stress-inducible Sdp1 Dual Specificity Phosphatase

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