Interaction between the Noncatalytic Region of Sid1p Kinase and Cdc14p Is Required for Full Catalytic Activity and Localization of Sid1p*

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Sid1p is a group II p21-activated kinase/germinal center kinase family member that is part of a signaling network required for cytokinesis in fission yeast. Germinal center kinases are characterized by well conserved amino-terminal catalytic domains followed by less conserved carboxy-terminal domains. The carboxy-terminal domain of group I germinal center kinases is moderately conserved and thought to be regulatory regions. Little is known about the carboxy-terminal domain of group II family members. Sid1p has been shown to bind the novel protein Cdc14p; however, the functional significance of this interaction is unknown. Here we report that the carboxy-terminal domain of Sid1p is an essential regulatory region. Our results indicate that this region contains the binding domain for Cdc14p, and that association is required for full Sid1p catalytic activity as well as intracellular localization. Furthermore, overexpression of the carboxy-terminal domain of Sid1p alone compromises the signaling of cytokinesis. We conclude that Cdc14p positively regulates the Sid1p kinase by binding the noncatalytic carboxy-terminal region of the protein.

The PAK1 family of protein kinases encompasses a large group of enzymes that have diverse functions including cell morphogenesis and stress response (1). PAKs typically have a carboxy-terminal Ser/Thr kinase domain preceded by an amino-terminal regulatory domain. The GCK family is a subfamily of PAKs that have an amino-terminal catalytic domain related to PAKs and a carboxy-terminal regulatory domain (1, 2). GCKs can be separated into two groups. Group I GCKs are most similar to the germinal center kinase, and all have homologous carboxy-terminal domains containing at least two PEST motifs, two binding sites for proteins with SH3 domains, and an additional conserved amino acid stretch. In contrast, little is known about Group II GCKs, which include human Ste20-like kinases Mst1 and Mst3 (3, 4), human oxidant stress-activated Ste20-like kinases Mst2 (5), and Dicyostelium discoideum severin kinase (6). In general, Group II GCKs share significant homology with Group I GCKs in the catalytic domain; however, their carboxy-terminal domains differ significantly from group I GCKs and are poorly understood. Biochemical evidence suggests that the carboxy-terminal region of some group II GCKs may contain an autoinhibitory domain (3, 5).

Sid1p is a group II GCK required for cytokinesis in the fission yeast, Schizosaccharomyces pombe (7). Like other GCKs, Sid1p has an amino-terminal catalytic region containing the 11 conserved domains typical of PAK/GCKs. In addition, Sid1p contains 200 amino acids carboxy-terminal to the catalytic domain. No function has yet been attributed to this region. Cdc14p, a novel protein with previously unknown function, binds Sid1p throughout the cell cycle, and together they function as an intermediate component in a signaling pathway termed the septation initiation network (SIN). The SIN provides temporal and spatial regulation of cell division by signaling the initiation of cytokinesis once mitosis has completed (8, 9). A key aspect of Sid1p/Cdc14p regulation is their temporal intracellular localization pattern; Sid1p/Cdc14p localizes asymmetrically to one spindle pole body (SPB) at the end of anaphase just prior to cytokinesis and leaves the SPB once septation completes (7). Genetic studies indicate that Sid1p and Cdc14p require each other to localize; however, the functional role of Cdc14p in the SIN is unknown.

In this study, we performed a structure/function analysis of Sid1p. We found that the carboxy-terminal domain of Sid1p is required to bind Cdc14p and promote the full catalytic activity of the kinase as well as to mediate the intracellular localization pattern of the protein.

EXPERIMENTAL PROCEDURES

Yeast Methods, Plasmids, and Sequencing—All S. pombe strains and methods were as described in Ref. 7. sid1 constructs were generated by PCR using the following oligonucleotide primers: Sid1N 5′-GGGG- GATCCGATGATCCTACTGATCTCAAT-3′; Sid1N 5′-GGGGGAT- CCCTAGTCTTTCACAGTTGTGC-3′; Sid1C 5′-GGGGGATCCG- ATTATCCCTTGTGTGCAG-3′; Sid1C 5′-GGGGGATCCCTTC- CATTTAGACTCTTTCTTCTT-3′; and Sid1 C-trunc, 5′-GGGGGATGCC- CTAGTTGTCGATAGATGGTAAGTTTGTAAC-3′ (Integrated DNA Technologies). Each oligonucleotide incorporated 5′ and 3′ BamHI sites (underlined). Sid1 expresses the full 1478 nucleotides of the sid1 gene, whereas Sid1N and Sid1C correspond to nucleotides 1–800 and 801–1458, respectively. Sid1ΔC corresponds to nucleotides 1–1371. DNA products were subcloned into the BamHI site of pRep41HA or pRep42GFP vectors and transformed into S. pombe strains by electroporation. These vectors express inserts from the inducible weak nmt1 promoter in the absence of thiamine and incorporate either an amino-terminal 3× HA or a GFP epitope tag (10, 11). For rescue experiments, three (+) signs were assigned as a measure of wild-type rescue, and a (−) was assigned to no rescue. (+) was assigned to Sid1N and Sid1ΔC because most cells did not survive; however, a few cells could survive and form colonies but clearly did not exhibit wild-type growth kinetics. Because the pRep41HA vector contains a leu gene and thus cannot be...
used in the 13myc-cdc14::leu1+ strain, co-immunoprecipitation studies used the ura4+ -based pREP42GFP expression vector. GFP-Sid1p is functionally equivalent to its HA-tagged counterpart (data not shown). GFP-Sid1K38R was constructed by site-directed mutagenesis with QuikChange (Stratagene) using pREP42GFP-Sid1 as the template. The mutant was confirmed to be nonfunctional by plasmid rescue of sid1–125. For overexpression studies, BamHI fragments of sid1N and sid1C were excised from pREP41HA vectors and subcloned into the BamHI site of pREP1. pREP1 expresses the inserts from the full strength nmt1 promoter (which is ~12× stronger than the weak (pREP41/42) nmt1 promoter (11). In all cases, plasmid-based expression was induced in S. pombe cells three times in thiamine-replete medium and transferring to medium lacking thiamine. For the sequencing of sid1–125 and sid1–239, genomic DNA of the mutants was prepared, and the sid1 locus was amplified by PCR and sequenced by the University of Massachusetts Medical School sequencing facility.

Co-immunoprecipitation Experiments—GFP-Sid1p constructs were induced in 13myc-cdc14 cells, and cells were grown to mid-log phase. Equal numbers of cells were harvested by centrifugation and lysed by vortexing the cell pellets with 425–600-μm acid-washed glass beads (Sigma). The protein lysate was removed from the glass beads by rinsing two times with 750 μl of Nonidet P-40 lysis buffer (1% Nonidet P-40, 150 mM NaCl, 2 mM EDTA, 6 mM Na2HPO4, and 4 mM NaH2PO4 plus protease inhibitors), and the lysates were cleared by centrifugation at 14,000 rpm for 10 min. The lysates were then split into two equal volumes, and 1 μl of a noncommercial GFP antibody (a gift of Pam Silver) or 1 μl of a noncommercial Myc antibody (a gift of Kathy Gould) was added. Antibodies were allowed to bind for 1 h at 4 °C while rocking. 40 μl of a 50:50 slurry of protein G beads (Sigma) and Nonidet P-40 buffer was then added to each lysate and allowed to bind for 1 h at 4 °C while rocking. Protein G beads were washed three times with Nonidet P-40 lysis buffer, 30 μl of 2× SDS sample buffer was then added to each tube, and samples were boiled for 2 min and centrifuged for 5 min at 14,000 rpm. Immunopurified proteins were resolved by SDS-PAGE on 10% gels and transferred to polyvinylidene difluoride membranes (Millipore) for Western blot analysis. Membranes were probed with anti-GFP (1:1000) or anti-Myc (1:2000) (gifts of Kathy Gould).

In Vitro Transcription/Translation—HA-tagged sid1 and cdc14 cDNAs were amplified from pREP41HA expression vectors by PCR using the 5′ oligonucleotide primer 5′-CCCTAATACGACTCACTATA-GGGGCTCGAGCCCATGGCATACCCTTAC-3′ (incorporated the T7 promoter (underlined) 5′ to the HA tag (Integrated DNA Technologies) and the 3′ primers 5′-GGGGGATCCCTATCCATTTAGACTTCGGATACAGTCTTCGAGCT-3′ and 5′-GGGGGATCCCTATCTTCCTTACGAAAGGTTGATGG-3′ which incorporated a stop codon for sid1 and cdc14, respectively (Integrated DNA Technologies). PCR products were cleaned and concentrated to 30 μl with Qiagen columns, and 2 μl of DNA was then added to a 25-μl TNT®-coupled reticulocyte lysate system (Promega). In addition, each reaction contained 12.5 μl of reticulocyte lysate, 1 μl of TNT® reaction buffer, 0.5 μl of TNT® T7-ppRNA polymerase, 0.5 μl of ampicillin acid mixture (minus 1 mM methionine), 1 μl of Redivue l-[35S]methionine (Amersham Pharmacia Biotech), 0.5 μl (2 units) of RNasin (Promega), and 7.0 μl of nuclease-free water according to the manufacturer’s protocol. Reactions were incubated at 30 °C for 90 min and then diluted to 0.5 ml with Nonidet P-40 buffer. For characterizing the catalytic role of Cdc14p, HA-Sid1 and HA-Cdc14 were made in duplicate, and one lysate of each was pooled with the other. A luciferase reaction (DNA provided by the manufacturer) was also carried out as a control. Reactions were then analyzed for kinase activity as described below. To confirm that equal amounts of protein were expressed in each reaction, 5 μl was removed prior to immunoprecipitation and boiled 2 min in 20 μl of 2× SDS sample buffer, and the proteins were separated by SDS-PAGE. Gels were processed for fluorography using Amplify (Amersham Pharmacia Biotech) according to the manufacturer’s protocol.

Kinase Assays—Kinase assays are described in (7). For analyzing kinase activity of the Sid1 constructs, HA-tagged Sid1 proteins were induced in S. pombe cells from the pREP41HA vector as described above. Lysates were prepared and subjected to immunoprecipitation with 1 μl of HA (12CA5) antibody (BAbCO) and protein G beads as described above. Protein G beads were further washed with kinase assay buffer (100 mM MgCl2,100 mM Tris, pH 7.4), and then each reaction was resuspended in 30 μl of kinase assay buffer containing 10 μg of myelin basic protein (Sigma), 50 μM ATP, and 0.5 μCi of Redivue l-[35S]ATP (Amersham Pharmacia Biotech). Reactions were incubated at 30 °C for 30 min with frequent mixing. Reactions were stopped by the addition of 20 μl of 2× SDS sample buffer and boiling for 2 min. Samples were cleared by centrifugation for 5 min at 14,000 rpm, and products were resolved by SDS-PAGE on 15% gels. Gels were dried and exposed to a PhosphorImager screen, and quantitative analysis was performed using Molecular Dynamics software. To monitor the protein level, proteins were resolved on 10% SDS-polyacrylamide gels, and Western blot analysis was performed as described above with HA antibody (12CA5–1:1000). The percent kinase activity was determined by calculating the relative activity compared with full-length Sid1, and the percent activity was corrected for protein level using a Bio-Rad Model GS-700 Imaging Densitometer and Multi-Analyst Software. For kinase assays of in vitro translation products, the procedure was the same except that diluted reticulocyte lysates were used as the source of protein (described above). The percent kinase activity was determined by calculating the activity relative to background. In all cases, kinase activity is expressed as the mean ± the range.

Microscopy Experiments—Fluorescence microscopy was performed as described in Ref. 7. S. pombe cells carrying GFP-Sid1 constructs were grown in thiamine-containing medium and then fixed by suspending in 100% methanol at −20 °C for 8 min followed by two washes in phos-
in wild-type SDS-PAGE, transferred to a polyvinylidene difluoride membrane, and probed with HA antibody. Growth to mid-log phase, and tagged proteins were immunoprecipitated from cell lysates with HA antibody. Immunoprecipitates were separated by SDS-PAGE, fixed, and subjected to kinase assays using myelin basic protein as an artificial substrate. Kinase activity was normalized to protein level in each experiment, and the percent relative kinase activity compared with full-length Sid1p is expressed as the mean ± the range.

**RESULTS**

To determine whether the carboxyl-terminal nonkinase portion of Sid1p is important for Sid1p function, we created HA-tagged Sid1 constructs to express full-length Sid1 protein, the amino-terminal kinase domain (Sid1N), and carboxyl terminus (Sid1C). The carboxyl-terminal epitope-tagged Sid1p is non-functional. Therefore we also deleted the extreme carboxyl terminus of Sid1p (Sid1ΔC) to determine whether this region is crucial for function (Fig. 1A). To determine whether the constructs were functional in vivo, HA-Sid1 constructs were tested for their ability to rescue *sid1*Δ−125 and *sid1*Δ−239 temperature-sensitive cells at a restrictive temperature (36 °C) when induced. *sid1*Δ−125 contains a single amino acid change (L140P) in domain VI of the kinase domain (this study). *sid1*Δ−239, a weaker allele, contains a single change (L12P) just upstream of domain I of the kinase domain (this study). Full-length HA-Sid1 rescued both alleles, whereas HA-vector and HA-Sid1C did not (Table I). HA-Sid1N and HA-Sid1ΔC did not rescue the *sid1*Δ−125 strain; however, they slightly rescued *sid1*Δ−239. We also examined the ability of each construct to rescue a *sid1*Δ deletion strain (*sid1Δ*::ura), and the results were identical to those with *sid1*Δ−125. These data indicate that the carboxyl terminus is required for the essential function of Sid1p.

Because the Sid1p carboxyl terminus is essential for viability, we next sought to identify its function. Sid1p is in a complex with Cdc14p (7); therefore we first tested whether the carboxyl terminus of Sid1p might mediate this interaction by co-immunoprecipitation experiments. To facilitate these studies, amino-terminal GFP-tagged versions of the gene constructs were expressed in *S. pombe* cells that contain 13myc-cdc14. 13Myc-Cdc14p was detected in GFP immunoprecipitates from cells expressing GFP-Sid1 and GFP-Sid1C but not GFP, GFP-Sid1N, or GFP-Sid1ΔC (Fig. 1B, middle). To control for 13Myc-Cdc14p expression, a Myc immunoprecipitate and immunoblot were performed (Fig. 1B, bottom). To control for GFP-Sid1p expression, a GFP immunoprecipitate and immunoblot were performed (Fig. 1B, top). These data indicate that the carboxyl-terminal portion of Sid1p binds Cdc14p.

Some evidence suggests that the carboxyl terminus of group II GCKs contain an autoinhibitory domain (3, 5). Therefore we next examined the kinase activity of the HA-Sid1 constructs immunopurified from *S. pombe* cell lysates to determine whether removal of the carboxyl terminus stimulated kinase activity. The expression of HA-Sid1 and the corresponding constructs was confirmed by Western blot analysis. (Fig. 2A). Relative kinase activity was high for HA-Sid1 immune complexes and barely above background for vector alone and Sid1C (Fig. 2B). HA-Sid1N immune com-

### Footnotes

1. D. McCollum, unpublished observations.
complexes, which are deleted for the carboxyl-terminal domain, exhibited a significant reduction in kinase activity (~40% of Sid1). HA-Sid1ΔC immune complexes also had reduced kinase activity, though not as pronounced as Sid1N. These data show that the carboxyl noncatalytic region of Sid1p does not suppress activity but rather is important for the full activity of the kinase in vivo.

Deletion of the entire carboxyl terminus or extreme carboxyl terminus of Sid1p results in decreased kinase activity. Because the carboxyl terminus contains the Cdc14p binding domain, we wondered whether Cdc14p was required for full Sid1p catalytic activity in vitro. To test this, we generated HA-tagged versions of Sid1p and Cdc14p by in vitro transcription/translation (Fig. 2C), immunopurified the products alone or in combination, and analyzed them for kinase activity (Fig. 2D). No DNA, HA-Cdc14, and Luciferase exhibited only background levels of activity (Fig. 2D). In contrast, HA-Sid1p reproducibly exhibited a moderate level of kinase activity, which increased in the presence of equal amounts of HA-Cdc14p. These data indicate that Cdc14p is required for full Sid1p catalytic activity in vitro.

Genetic studies indicate that asymmetric SPB localization of Sid1p/Cdc14p in late anaphase requires Cdc14p function (7). Because Cdc14p is required for full Sid1p catalytic activity, we tested whether both physical interaction between Sid1p and Cdc14p and Sid1p kinase activity were required for Sid1p SPB localization. We first tested whether Sid1 proteins that lack the ability to bind Cdc14p can still localize. Full-length GFP-Sid1 as well as GFP-Sid1C were detected at SPBs in nearly 100% of late anaphase cells (Fig. 3, panels A and C, respectively), whereas GFP-Sid1N, GFP-Sid1ΔC, and GFP alone gave no SPB signal (Fig. 3, panels B, D, and F, respectively). These results indicate that the extreme carboxyl terminus of Sid1p, which is required for Cdc14p binding, is also required for SPB localization.

The fact that GFP-Sid1C alone can localize to the SPB in the absence of a kinase domain strongly suggests that Cdc14p does not promote Sid1p localization by enhancing Sid1p kinase activity. To confirm this observation, we examined the localization of GFP-Sid1p (kinase-dead), which has a conserved lysine to arginine mutation (K38R) in domain I of the kinase domain (7). Consistent with kinase activity not being required for localization, GFP-Sid1K38R localized efficiently to one SPB at the end of anaphase (Fig. 3, panel E).

SIN proteins are essential for viability, and mutants exhibit a cytokinesis defect, becoming long and multinucleate and eventually lysing (13). Sid1p requires Cdc14p for kinase activity and localization. Therefore we reasoned that high expression of the carboxyl-terminal fragment of Sid1p containing the Cdc14p binding domain might titrate Cdc14p from Sid1p and elicit a SIN phenotype. DNA staining indicates that high expression of the carboxyl-terminal fragment of Sid1p containing the Cdc14p binding domain might titrate Cdc14p from Sid1p and elicit a SIN phenotype. DNA staining indicates that high expression of Sid1C induces a SIN phenotype marked by long multinucleate cells (Fig. 4A, panel 1) compared with cells expressing no insert (Fig. 4A, panel 1) or Sid1N (Fig. 4A, panel 2), which show normal division patterns. All cells grown in noninducing conditions as well as cells overproducing full-length Sid1p divide normally (data not shown). Cdc7p functions upstream of Sid1p in the SIN and localizes to the SPB just prior to Sid1p. The induction of Sid1C in strains that expressed chromosomal GFP-tagged versions of Cdc7p or Sid1p indicated that high expression of Sid1C specifically disrupts GFP-Sid1p localization (Fig. 4B, panels 3 and 4) but not Cdc7p-GFP (Fig. 4B, panels 1 and 2).

**DISCUSSION**

Sid1p is a group II PAK/GCK that binds Cdc14p and is required for cytokinesis in fission yeast (7). Group II GCKs have an amino-terminal kinase domain and a carboxyl-terminal domain thought to be a regulatory region (1). In the case of Sid1p, we find that the carboxyl noncatalytic region of Sid1p contains the binding domain for Cdc14p, which is required for full Sid1p catalytic activity and intracellular localization. Thus the carboxyl terminus of Sid1p appears to have an important role in regulating Sid1p kinase activity and localization through binding Cdc14p. We favor a model in which Cdc14p binds the carboxyl terminus of Sid1p, and this alters the conformation of the carboxyl terminus such that it can be in a position to allow full activation of Sid1p kinase activity. This conformation is competent to localize to SPBs when signaled during late anaphase.

**FIG. 3. Localization of Sid1p constructs.** GFP-Sid1 constructs were expressed in wild-type *S. pombe* cells, fixed, and examined by fluorescence microscopy. GFP fluorescence and corresponding DNA staining with DAPI for GFP-Sid1 (A), GFP-Sid1N (B), GFP-Sid1C (C), GFP-Sid1ΔC (D), and GFP-Sid1K38R (E) are shown. A, control, GFP alone was analyzed (F).

**FIG. 4. High expression of Sid1C perturbs cytokinesis.** A, vector alone (1), Sid1N (2), or Sid1C (3) were overexpressed in wild-type *S. pombe* cells from the full strength *nmt1* promoter (see “Experimental Procedures”). Following induction, cells were fixed and processed for fluorescence microscopy. DAPI staining of DNA is shown. B, Sid1C was repressed (+T, 1 and 3) or induced from the full strength *nmt1* promoter (−T, 2 and 4) in *S. pombe* cells that also expressed Cdc7p-GFP or GFP-Sid1p from the endogenous locus. GFP fluorescence and DAPI staining of DNA are shown.
Some GCK family members have autoinhibitory domains in their noncatalytic regulatory regions. It is possible that the function of sid1C is to bind a Sid1p autoinhibitory domain in the carboxyl terminus and relieve inhibition. However, we feel this is unlikely because the deletion of the carboxyl terminus or extreme carboxyl terminus of Sid1p does not stimulate the kinase activity of the protein when purified from S. pombe lysates. The observation that sid1C exhibits higher kinase activity than Sid1N may result from other endogenous factors that co-purify with and help to stabilize the protein.

We observed that Sid1N and Sid1ΔC slightly rescue the weak sid1–239 allele, which contains a single amino acid change just outside the kinase domain, but not a sid1–125 allele, which contains a mutation within the kinase domain. One possible explanation of why Sid1N and Sid1ΔC can rescue sid1–239 but not sid1–125 is that dimerization could occur between the weak mutant Sid1p and the kinase domain fragment, forming a functional enzyme, whereas the more severe mutation cannot be compensated for. It has been reported that some group II GCKs homodimerize (3); however, we have been unable to detect homodimerization of Sid1p (data not shown). Because the sid1–239 allele is significantly weaker than sid1–125, we suspect that simply overproducing the slightly active sid1N and sid1ΔC gene products with a slightly active sid1–239 gene product can result in synergistic complementation. The fact that sid1–239 contains a single point mutation outside of the kinase domain supports this idea.

Cdc14p is a novel protein with no significant homology to any other known protein (14). Sid1p is in a complex with Cdc14p throughout the cell cycle, and the complex exhibits a modest peak in kinase activity around septation (7), but until now little has been known about the biochemical role of Cdc14p in the SIN. Our observation that the domain in Sid1p that binds Cdc14p is also required for localization suggests that physical association of Cdc14p with the Sid1p carboxyl terminus is required for localization. Because both Sid1p and Cdc14p require each other to localize (7), it is possible that each subunit contacts an SPB component independently but only when both subunits are present. We also observe that Cdc14p is required for full Sid1p catalytic activity in vitro. Cdc14p may simply be required to structurally hold Sid1p in a conformation that can fully be activated. In addition, Cdc14p could provide substrate recognition information in vivo. Because Cdc14p is required for both full Sid1p catalytic activity and localization, it is interesting that Sid1p kinase activity is not required for localization in vivo. Perhaps Cdc14p has dual functions: one in maintaining Sid1p kinase in a state that can be fully active and another in contributing to SPB localization and/or substrate recognition.

In this study, we have not only identified the carboxyl terminus of Sid1p as a regulatory domain required to bind Cdc14p but also characterized the previously unknown crucial role of Cdc14p in regulating both Sid1p kinase activity and localization. It will be interesting in future studies to determine whether the activation of Sid1p kinase activity and localization of the Sid1p/Cdc14p complex are mediated directly through Cdc14p or whether Cdc14p simply serves as a companion to Sid1p required to maintain Sid1p in a fully functional state. Furthermore, Cdc14p is one of the first proteins known to interact with the carboxyl terminus of a group II GCK. It will be interesting to determine whether the carboxyl termini of other group II GCKs bind to proteins involved in the localization and activation of the kinase.

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