Cse1p Is Required for Export of Srp1p/Importin-α from the Nucleus in *Saccharomyces cerevisiae*  

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In metazoan cells, the CAS protein has been shown to function as a recycling factor for the importin-α subunit of the classical nuclear localization signal receptor, exporting importin-α from the nucleus to allow its participation in multiple rounds of nuclear import. CAS is a member of a family of proteins that bear homology to the larger subunit of the nuclear localization signal receptor, importin-α, and that are found in all eukaryotes from yeast to humans. Sequence similarity identifies the product of the *Saccharomyces cerevisiae CSE1* gene as a potential CAS homologue. Here we present evidence that Cse1p is the functional homologue of CAS: Cse1p is required to prevent accumulation of Srp1p/importin-α in the nucleus, it localizes to the nuclear envelope in a pattern typical of nuclear transport receptors, and it associates in vivo with Srp1p in a nucleotide-specific manner. We show further that mutations in CSE1 and *SRP1* have specific effects on their association and on the intracellular localization of Cse1p.

Bi-directional transport of macromolecules through nuclear pore complexes (NPCs) is an energy-dependent process that involves substrate recognition by a soluble transport receptor, binding of the receptor to nuclear pore proteins, and subsequent translocation of the receptor-cargo complex through the NPC. All transport receptors identified thus far are members of a family of proteins with similarity to the importin-β subunit of the dimeric classical nuclear localization signal (NLS) receptor (Refs. 1–3 and reviewed in Refs. 4 and 5). Importin-β family members are most homologous in their N termini (3), which contain a conserved domain that binds to the small GTPase Ran, an essential component of the transport machinery (6, 7). Proteins containing basic NLSs of the SV40 or bipartite variety bind to importin-α, which in turn binds to importin-β, serving as an adapter between these import substrates and their transport receptor (8–10).

After crossing the NPC, the importin-α/β dimer dissociates due to the interaction of importin-β with Ran-GTP instead of Ran-GDP, thus leaving importin-α free in the nucleus (11, 12).

Recently, the importin-β-like protein CAS (cellular apoptosis susceptibility protein) from Xenopus and HeLa cells has been identified as a recycling factor for importin-α that mediates export of the protein from the nucleus to allow its participation in further rounds of import (13).

The high degree of conservation of nuclear transport pathways between yeast and higher eukaryotes, combined with the genetic tractability of yeast, has made *Saccharomyces cerevisiae* a valuable tool for the identification of new transport factors as well as the elucidation of functional interactions between known factors (reviewed in Ref. 14). A search of the completed *S. cerevisiae* genome identified CSE1 as the potential yeast homologue of the CAS gene. The predicted protein products of the two genes are 40% identical (13). *CSE1* is an essential gene that was originally recovered in a screen for mutations that affect chromosome segregation (15). *SRP1*, the gene that encodes yeast importin-α (10, 16), was found to be a high copy suppressor of the cold-sensitive cse1-1 mutation (15). Here we show that Cse1p is required for export of Srp1p from the nucleus and that the two proteins interact in a GTP-dependent manner, as expected for a receptor-substrate export complex.

**EXPERIMENTAL PROCEDURES**

*Yeast Strains and Plasmids—*The *cse1-1* strain was a gift from M. Fitzgerald-Hayes (University of Massachusetts, Worcester, MA), and the *rat3-1* strain was from C. Cole (Dartmouth Medical School). The *srp1-31* and *srp1-49* strains have been previously published (17), as have *prp20-1* and *rna1-1* (47, 48). To integrate *CSE1* tagged with GFP at the C terminus, a 2.7-kilobase pair *PatI*KpnI fragment of *psP1536 (CEN TRP CSE1-GFP (18)) lacking the promoter and first 954 coding base pairs of *CSE1* was cloned into pRS314, generating *psP1610*. This plasmid was then linearized by *BstEII* digestion and was transformed into *S. cerevisiae* strains as for the Cse1-GFP strain. The *galactose-induced GFP-Crm1/Xpo1-GFP* strain was from T. Taura and M. Damelin, respectively, using similar strategies as for the Cse1-GFP strain. The galactose-induced GFP-Bcl2 fusion protein was produced from *psP1626*. *Immunofluorescence—*Srplp was localized in *CSE1* and *cse1-1* strains at 30 and 15 °C (12-h shift) using indirect immunofluorescence microscopy. Cells were fixed in 3.7% formaldehyde for 30 min, washed in 0.1 M potassium phosphate buffer, pH 6.5, and resuspended in P solution (0.1 M potassium phosphate buffer, pH 6.5, 1.2 M sorbitol). Cell walls were digested by incubation at 30 °C with 0.3 mg/ml zymolyase, 25 mM dithiothreitol, and cells were applied to slides precoated with 0.3% polylysine. Cells were permeabilized by treatment with 0.5% Nonidet P-40 in P solution and blocked with 1 mg/ml bovine serum albumin in PBS. Rabbit polyclonal anti-importin-α antibodies (gift from
D. Görlich, Zentrum für Molekulare Biologie der Universität Heidelberg, Germany) were used at a dilution of 1:1000 overnight. Fluorescein isothiocyanate-conjugated anti-rabbit secondary antibodies (Jackson ImmunoResearch) were used at 1:1000; 4',6-diamidino-2-phenylindole (1 μg/ml) was used to stain DNA.

**Anti-Cpe1p Antibodies**—To make a GST-Cpe1p expression plasmid, the CSE1 open reading frame was PCR amplified, and BamHI and XhoI sites were introduced at the ends using Vent polymerase with primers JKH38 (5'-CGGGATCCATGTCGAGTTTGGAAACGGTAGCC-3') and JKH31 (5'-CCGCTCGAGTTAATCAATAATTG-3'). The PCR product was digested with BamHI and AvaI and was cloned into the BamHI/XhoI sites of pGEX-4T-1 (Amersham Pharmacia Biotech) to generate pPS1611. GST-Cpe1p was expressed from pPS1611 in *Escherichia coli* by 3 h isopropyl-1-thio-β-D-galactopyranoside (0.1 mM) induction at 30 °C and was purified by binding to glutathione-Sepharose (Amersham Pharmacia Biotech) and elution with 50 mM Tris, pH 8.0, 5 mM reduced glutathione. Rabbit polyclonal antibodies were raised against full-length GST-Cpe1p (Covance Research Products, Inc.). The antibody was used at a 1:5000 dilution for immunoblotting.

**Immunoprecipitations**—100-ml cultures of Cse1-GFP or cse1-1-GFP strains were grown in rich medium to approximately 2 × 10^8 cells/ml and then were pelleted and washed once with water. Cells were lysed in 250 μl of ice-cold PBSMT (2 mM MgCl_2, 1 mM EDTA, 0.05% Triton X-100 in PBS) plus protease inhibitors (0.5 mM phenylmethylsulfonylfluoride, 3 μg/ml pepstatin A, leupeptin, aprotinin, and chymostatin) using glass beads in a FastPrep bead beater (Savant). After lysis, 500 μl more of PBSMT were added, and lysates were clarified by two 10-min microcentrifuge spins at 4 °C. Anti-GFP beads were prepared by incubating 300 μl of IgG-Sepharose (Amersham Pharmacia Biotech, 50% slurry in PBS) with 100 μl 1 mg/ml rabbit polyclonal anti-GFP antibodies (gift of J. Kahana) for 15 min on a room temperature rocker. Beads were washed three times with PBS, ending with a 50% slurry. 10 μl of anti-GFP beads were used to immunoprecipitate GFP-tagged proteins from 250 μl of lyase (approximately 1 mg/ml total protein concentration by Bio-Rad protein assay) by rocking at 4 °C for 1 h. The GST analogue GMPPNP (Sigma) was added to some lysates along with the beads (1 mM GMPPNP/2 mM MgCl_2). Beads were washed four times with ice-cold PBSMT and once with 500 μl of ice-cold PBSM (2 mM MgCl_2, 1 mM EDTA in PBS). Samples were prepared for gel analysis by adding 50 μl of 1X SDS sample buffer and boiling for 2 min.

**Immunoblotting**—Anti-GFP immunoprecipitates were resolved in 8 or 10% SDS-polyacrylamide gels (19), and proteins were transferred to nitrocellulose membranes using standard techniques (20). Anti-GFP antibodies were used at a 1:4000 dilution, and anti-importin-α antibodies were used at a 1:2000 dilution. Horseradish peroxidase-conjugated anti-rabbit secondary antibodies (Jackson ImmunoResearch) were used at a 1:5000 dilution for immunoblotting. Anti-GFP beads were used to localize Srp1p in wild type and cse1-1 cells at 30 °C (top panels) or after a 12-h shift at 15 °C (bottom panels). 4',6-Diamidino-2-phenylindole (DAPI) staining shows nuclei, and Nomarski shows phase images of cells.

**RESULTS AND DISCUSSION**

By amino acid sequence similarity, Cse1p is the closest CAS protein homologue in *S. cerevisiae*. We sought to determine whether it is also a functional homologue, that is, whether Cse1p is the recycling factor that returns Srp1p to the cytoplasm after it enters the nucleus along with a classical NLS-containing substrate. To address this question, we first examined the localization of Srp1p in the cold-sensitive *cse1-1* strain by direct immunofluorescence on wild type cells and *cse1-1* mutants. Anti-Srp1p staining shows a diffuse pattern throughout the cell (Fig. 1A), indicative of its continued movement in and out of the nucleus. At 15 °C, some wild type cells show a partial accumulation of Srp1p in the nucleus, suggesting that export is slowed in the cold. However, they still retain significant cytoplasmic Srp1p signal (Fig. 1G). In contrast, even at 30 °C, *cse1-1* cells show significant accumulation of Srp1p in the nucleus (Fig. 1D); at the nonpermissive temperature of 15 °C, anti-Srp1p signal is restricted to the nucleus in virtually 100% of the cells (Fig. 1J). This result implies that Cse1p plays a role in the nuclear export of Srp1p. Although *cse1-1* cells show nuclear accumulation of Srp1p even at the permissive temperature, they do not grow significantly slower than wild type cells at 30 °C (data not shown). This apparent contradiction may be explained by the fact that Srp1p appears to be a very highly expressed protein. Thus, even if a majority of the Srp1p in a cell is nuclear at the permissive temperature, its rate of recycling to the cytoplasm may be sufficient to allow nearly normal rates of nuclear import and population doubling.

To gain further evidence that Cse1p is the export receptor for Srp1p, we localized Cse1p in strains in which the endogenous *CSE1* or *cse1-1* locus was replaced with sequence encoding Cse1p with GFP fused to its C terminus. The integration strategy was such that the fusion protein is the sole form of Cse1p expressed in these strains (see “Experimental Procedures”). Fig. 2 shows the endogenous Cse1p and *cse1-1* proteins detected by immunoblotting with anti-Cse1p antibodies (lanes 1 and 3) and their respective GFP fusion proteins visualized by blotting with anti-GFP (lanes 2 and 4) or anti-GFP (lanes 6 and 8) antibodies. The predicted molecular masses of endogenous Cse1p/cse1-1 and the GFP fusion proteins are 109 and 135 kDa, respectively. Cse1p migrates slightly faster than expected on a 10% SDS-polyacrylamide gel, running closer to the 100-kDa marker than the 110-kDa marker (Fig. 2, lanes 1 and 3). There is an approximately 115-kDa protein that also reacts with the anti-Cse1p antibody in all lysates (Fig. 2, lanes 1–4). We do not know the identity of this band, but it is a formal possibility that it could be another importin-β-like protein. The sequenced yeast genome predicts at least two potential homologues, Lph2p and YGL241W, which would migrate at this approximate molecular size. The integrated Cse1p-GFP strain showed no growth defects compared with its parental strain (data not shown), indicating that the fusion protein was completely functional. Furthermore, when Cse1p-GFP was integrated into *cse1-1*, the resulting strain retained its cold sensitivity (data not shown), confirming that the integration strategy did not eliminate the original *cse1-1* mutation.

The wild type and mutant fusion proteins were localized by

**Fig. 1. Srp1p accumulates in the nucleus in *cse1-1* cells.** Polyclonal anti-importin-α antibodies were used to localize Srp1p in wild type and *cse1-1* cells at 30 °C (top panels) or after a 12-h shift at 15 °C (bottom panels). 4',6-Diamidino-2-phenylindole (DAPI) staining shows nuclei, and Nomarski shows phase images of cells.
fluorescence microscopy. Cse1-GFP shows a strong punctate nuclear envelope localization (Fig. 3A, top panels), similar to that normally observed for NPC proteins (e.g. see Ref. 21). The association of Cse1-GFP with nuclear pores was confirmed by localizing the fusion protein in a rat3-1 strain. This strain has a defect in mRNA export at high temperatures and exhibits clustering of NPCs at one or a few regions of the nuclear envelope at all temperatures (22). As shown in Fig. 3B, Cse1-GFP also shows a clustering pattern in rat3-1 cells, indicating that the localization seen in wild type cells represents authentic NPC association.

Most of the yeast importin-β-like proteins examined thus far show a similar concentration at nuclear pores but with varying degrees of background cytoplasmic and nuclear localization (23–29). For Cse1-GFP, the non-pore-associated signal was essentially undetectable. The mutant cse1-1-GFP protein also localized to the nuclear envelope but showed higher intranuclear signal than the wild type fusion protein (Fig. 3A, lower panels) at both 30 and 15 °C, suggesting that the mutant protein cannot exit the nucleus as well as wild type Cse1p.

Similar intranuclear accumulation of Cse1-GFP was seen in a prp20-1 strain shifted to the nonpermissive temperature of 37 °C (Fig. 3C, middle panels). The PRP20 gene product is the guanine nucleotide exchange factor for Gsp1p (30, 31), which is the yeast Ran protein (32–34). prp20-1 cells exhibit both protein import and mRNA export defects (30, 31, 35, 36). The accumulation of Cse1-GFP inside the nucleus of prp20-1 cells is indicative of a requirement for Gsp1p-GTP for translocation of Cse1p through the NPC. We also localized Cse1-GFP in rna1-1 cells. RNA1 encodes the GTPase-activating protein for Gsp1p (37–39). The mutant rna1-1 protein fails to stimulate Gsp1p-GTP hydrolysis, and therefore rna1-1 cells have elevated levels of Gsp1p-GTP (39). In the rna1-1 strain, the NPC localization of Cse1-GFP was diminished, and the protein was localized throughout the cell (Fig. 3C, right panels). Because Srp1p shows a similar diffuse localization, this result may indicate that, in the presence of Gsp1p-GTP, Cse1p constitutively associates with its transport substrate.

We next looked for a biochemical interaction between Cse1p and Srp1p, which would be expected if the two form a genuine receptor-substrate export complex. Rabbit polyclonal anti-GFP antibodies bound to IgG-Sepharose beads were used to immunoprecipitate Cse1-GFP from lysates prepared from the integrated CSE1-GFP and cse1-1-GFP strains (Fig. 4A, top panel, lanes 4–9). Co-immunoprecipitation of Srp1p was assayed by immunoblotting with anti-importin-α antibodies (Fig. 4A, bottom panel, lanes 4–9). In the absence of any nucleotide analogue, Cse1-GFP immunoprecipitates contained only background levels of Srp1p, similar to a control in which beads were incubated with lysate that did not contain any GFP fusion protein (Fig. 4A, compare lanes 4 and 6). However, when the nonhydrolyzable GTP analogue GMPPNP (1 mM) was added to the Cse1-GFP lysate, co-immunoprecipitation of Srp1p was vastly stimulated (Fig. 4A, compare lanes 5 and 7). Srp1p did not co-immunoprecipitate with two other importin-β-like proteins expressed as C-terminal GFP fusions (Xpo1-GFP and Sxm1-GFP) or with a fusion of GFP to the N terminus of β-galactosidase (Fig. 4B, lanes 4–9). Therefore, the interaction between Cse1-GFP and Srp1p is specific; it does not reflect an affinity of Srp1p either for GFP or for multiple members of the importin-β family. The failure of Srp1p to interact with Crm1/Xpo1-GFP is especially significant because Crm1p/Xpo1p is also an export receptor, known to mediate export of proteins that contain leucine-rich nuclear export signals (40, 41).

The CAS protein has been shown to bind cooperatively to importin-α and RanGTP (13). Yeast cell lysates prepared under normal conditions would be expected to contain Gsp1p primarily in the GDP-bound state due to the GTPase activating activity of Rna1p. The addition of the nonhydrolyzable GTP analogue GMPPNP to these lysates should restore the GDP-bound population of Gsp1p. The fact that the Cse1-GFP/Srp1p interaction only occurs in the presence of GMPPNP implies that it represents a Gsp1p-GTP-dependent export complex.

Srp1p did not co-immunoprecipitate from lysates from cse1-1-GFP expressing cells even in the presence of GMPPNP (Fig. 4A, lane 9). This result suggests that the cse1-1 mutation affects the formation of the Cse1p-Srp1p export complex and provides an explanation for the previously observed suppression of cse1-1 by high copy SRP1 (15). The cse1-1 strain contains a mutation that changes amino acid residue 220 from
aspartate to asparagine (D220N). This residue lies outside the conserved N-terminal Ran binding motif that is shared by importin-β family members; however, the experimentally determined Ran binding domain of importin-β extends beyond this conserved region (42, 43). Therefore, it is possible that the cse1-1 mutation disrupts Gsp1p-GTP binding and thus interferes with the formation of a cooperative Cse1p-Srp1p-Gsp1p-GTP complex. Alternatively, the D220N mutation may identify a region of Cse1p that is directly involved in binding to Srp1p.

Cse1p-GFP was also integrated into two strains containing mutations in SRP1, srp1-31, and srp1-49, and similar immunoprecipitation experiments were carried out using lysates from these strains. Like wild type Srp1p, the mutant srp1-49 protein co-immunoprecipitated with Cse1p-GFP in the presence of GPPNP, but the srp1-31 protein did not (Fig. 4B, compare lanes 7 and 9). Note that the level of Cse1p-GFP expression in the srp1-31 strain was decreased relative to the wild type and srp1-49 strains but that longer exposures of the immunoblot still did not visualize any co-precipitating srp1-31 protein.

Srp1p contains a central domain consisting of eight 42-amino acid tandem repeats of the armadillo (arm) motif found in a family of proteins that includes the Drosophila armadillo protein and the vertebrate β-catenin and plakoglobin proteins (17). X-ray crystal structures have recently been determined for the arm repeats of β-catenin (44) and importin/karyopherin-α (45); both structures exhibit an extensive shallow surface groove that seems to function as a specific protein-protein interaction module. In the case of importin/karyopherin-α, the surface groove constitutes the binding site for positively charged NLS peptides (45). A conserved 41-amino acid domain not present in the crystallized protein has been identified as the importin-β binding domain (46).

The srp1-49 mutation lies within the first arm repeat (17), whereas the srp1-31 mutation falls just N-terminal to the start of the arm domain (17) and C-terminal to the importin-β binding domain. The different interaction properties of the two mutant Srp1 proteins may indicate that the srp1-31 mutation lies within a region of the protein that is required for binding to Cse1p, but further studies will be required to elucidate the precise domain in Srp1p that interacts with Cse1p. srp1-31 cells exhibit a strong nuclear import defect for classical NLS-containing substrates, whereas srp1-49 cells show a much weaker defect (16). This observation is consistent with the failure of the srp1-31 protein to bind to Cse1p, but paradoxically, the mutant protein does not mislocalize to the nucleus. This contradiction may be explained if the srp1-31 mutation abrogates binding to both Cse1p and importin-β. In this case, the mutant protein would never enter the nucleus and thus could not be trapped by a lack of interaction with its recycling factor.

The CAS protein has been shown to export importin-α in metazoan cells. Here we present evidence that Cse1p, the S. cerevisiae CAS homologue, is the export receptor for Srp1p, which is the yeast importin-α protein. We have demonstrated a biochemical interaction between Cse1p and Srp1p that requires the presence of GTP and have identified mutations in both Cse1p and Srp1p that abolish this interaction. The facility of genetic and biochemical analysis in yeast will make it an excellent system for further study of the importin-α recycling pathway.

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REFERENCES
1. Adam, E. J. H., and Adam, S. A. (1994) J. Cell Biol. 125, 547–555
2. Radu, A., Biebel, G., and Moore, M. S. (1995) Proc. Natl. Acad. Sci. U. S. A. 92, 1769–1773
3. Görlich, D., Dabowski, M., Bischoff, F. R., Kutz, U., Bork, P., Hartmann, E., Prehn, S., and Izaquirre, E. (1997) J. Cell Biol. 138, 65–80
4. Weis, K. (1998) Trends Biochem. Sci. 23, 185–189
5. Izaquirre, E., and Adam, S. (1998) RNA 4, 351–364
6. Moore, M. S., and Blobel, G. (1993) Nature 365, 661–663
7. Melchior, F., Paschal, B., Evans, J., and Gerace, L. (1993) J. Cell Biol. 123, 1649–1659
8. Görlich, D., Vogel, F., Mills, A. D., Hartmann, E., and Laskey, R. A. (1995) Nature 377, 246–248
9. Imamoto, N., Shimamoto, T., Takao, T., Tahibana, T., Kose, S., Matsubae, M., Sekimoto, T., Shimoshi, Y., and Yoned, Y. (1995) EMBO J. 14, 3617–3626
10. Moretina, I., Blobel, G., and Radu, A. (1995) Proc. Natl. Acad. Sci. U. S. A. 92, 2008–2011
11. Rexach, M., and Blobel, G. (1995) Cell 83, 683–692
12. Görlich, D., Panté, N., Kutz, U., Aebi, U., and Bischoff, F. (1996) EMBO J. 15, 5584–5594
13. Kutz, U., Bischoff, F., Kostka, S., Kraft, R., and Görlich, D. (1997) Cell 90, 1061–1071
14. Corbett, A. H., and Silver, P. A. (1997) Microbiol. Rev. 61, 193–211
15. Xiao, Z., McGrew, J. T., Schroeder, A. J., and Fitzgerald-Hayes, M. (1993) Mol. Cell. Biol. 13, 4691–4702
16. Loeb, J. D. L., Schlenstedt, G., Pelling, M., Kornitzer, D., Silver, P. A., and Fink, G. (1995) Proc. Natl. Acad. Sci. U. S. A. 92, 7647–7651
17. Yano, R., Oakes, M. L., Tabb, M. T., and Nomura, M. (1994) Proc. Natl. Acad. Sci. U. S. A. 91, 6880–6884
18. Taura, T., Krebber, H., and Silver, P. A. (1998) Proc. Natl. Acad. Sci. U. S. A. 95, 7427–7432
19. Laemmli, U. K. (1970) Nature 227, 680–685
20. Ausubel, F. M., Brent, R., Kingston, R. E., Moore, D. D., Seidman, J. G., Smith, J. A., and Struhl, K. (1987) Current Protocols in Molecular Biology (Chanda, V. B., ed) John Wiley & Sons, Inc., New York
21. Davis, L. I., and Fink, G. R. (1990) Cell 61, 965–978
22. Li, O., Heath, C. V., Amberg, D. C., Dockendorff, T. C., Copeland, C. S., Snyder, M., and Cole, N. C. (1995) Mol. Biol. Cell 6, 401–417

2 A. Schroeder, personal communication.
3 J. K. Hood and P. A. Silver, unpublished results.
23. Aitchison, J. D., Blobel, G., and Rout, M. P. (1996) Science 274, 624–627
24. Koepp, D. M., Wong, D. H., Corbett, A. H., and Silver, P. A. (1996) J. Cell Biol. 133, 1163–1176
25. Simos, G., Tekotte, H., Grosjean, H., Segref, A., Sharma, K., Tollervey, D., and Hurt, E. C. (1996) EMBO J. 15, 2270–2284
26. Rout, M. P., Blobel, G., and Aitchison, J. D. (1997) Cell 89, 715–725
27. Schlenstedt, G., Smirnova, E., Deane, R., Solsbacher, J., Kutay, U., Görlich, D., Ponstingl, H., and Bischoff, F. (1997) EMBO J. 16, 6237–6249
28. Seedorf, M., and Silver, P. A. (1997) Proc. Natl. Acad. Sci. U. S. A. 94, 8590–8595
29. Ferrigno, P., Posas, F., Koepp, D., Saito, H., and Silver, P. A. (1998) Proc. Natl. Acad. Sci. U. S. A. 95, 5606–5614
30. Aebi, M., Clark, M. W., Vijayraghavan, U., and Abelson, J. (1990) Mol. Gen. Genet. 224, 72–80
31. Fleischmann, M., Clark, M. W., Forrester, W., Wickens, M., Nishimoto, T., and Aebi, M. (1991) Mol. Gen. Genet. 227, 417–423
32. Belhumeur, P., Lee, A., Tam, R., DiPaolo, T., Fortin, N., and Clark, M. W. (1993) Mol. Cell. Biol. 13, 2152–2161
33. Schlenstedt, G., Saavedra, C., Loeh, J. D., Cole, C. N., and Silver, P. A. (1995) Proc. Natl. Acad. Sci. U. S. A. 92, 225–229
34. Kadowaki, T., Goldfarb, D., Spitz, L. M., Tartakoff, A. M., and Ohno, M. (1993) EMBO J. 12, 2929–2937
35. Kadowaki, T., Zhao, Y., and Tartakoff, A. (1992) Proc. Natl. Acad. Sci. U. S. A. 89, 2312–2316
36. Amberg, D. C., Fleischmann, M., Stagljar, I., Cole, C. N., and Aebi, M. (1993) EMBO J. 12, 233–241
37. Becker, J., Melchior, F., Gerke, V., Bischoff, F. R., Ponstingl, H., and Wittinghofer, A. (1995) J. Biol. Chem. 270, 11860–11865
38. Bischoff, F. R., Krebber, H., Kempf, T., Hermes, I., and Ponstingl, H. (1995) Proc. Natl. Acad. Sci. U. S. A. 92, 1749–1753
39. Corbett, A. H., Koepp, D. M., Lee, M. S., Schlenstedt, G., Hopper, A. K., and Silver, P. A. (1995) J. Cell Biol. 130, 1017–1026
40. Stade, K., Ford, C., Guthrie, C., and Weiss, K. (1997) Cell 90, 1041–1050
41. Neville, M., Stutz, P., Lee, L., Davis, L. L., and Rosbash, M. (1997) Curr. Biol. 7, 767–775
42. Chi, N. C., Adam, E. J. H., and Adam, S. A. (1997) J. Biol. Chem. 272, 6818–6822
43. Kutay, U., Izzaurralde, E., Bischoff, F. R., Mattaj, I. W., and Görlich, D. (1997) EMBO J. 16, 1153–1163
44. Huber, A. H., Nelson, W. J., and Weiss, W. I. (1997) Cell 90, 871–882
45. Conti, E., Uy, M., Leighton, L., Blobel, G., and Kuriyan, J. (1998) Cell 94, 193–204
46. Görlich, D., Henklein, P., Laskey, R. A., and Hartmann, E. (1996) EMBO J. 15, 1810–1817
47. Atkinson, N. S., Dunst, R. W., and Hopper, A. K. (1985) Mol. Cell. Biol. 1, 907–915
48. Aebi, M., Clark, M. W., Vijayraghavan, U., and Abelson, J. (1990) Mol. Gen. Genet. 224, 72–80
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