Scaffolding proteins exist in eukaryotes to properly assemble signaling proteins into specific multimeric functional complexes. JLP is a novel leucine zipper protein belonging to a family of scaffolding proteins that assemble JNK signaling modules. JLP is a proline-rich protein that contains two leucine zipper domains and a highly conserved C-terminal domain. We have identified kinesin light chain 1 (KLC1) as a binding partner for the second leucine zipper domain of JLP using yeast two-hybrid screening. The interaction domain of KLC1 was mapped to its tetra tripeptide repeat, which contains a novel leucine zipper-like domain that is crucial for the interaction with JLP. Mutations of Leu-280, Leu-287, Val-294, and Leu-301 within this domain of KLC1 disrupted its ability to associate with JLP. Immunofluorescence studies showed that JLP and KLC1 co-localized in the cytoplasm and that the localization of JLP was dependent on its second leucine zinc. Ectopic expression of a dominant negative form of KLC1 resulted in the mislocalization of endogenous JLP. Moreover, the association between JLP and KLC1 occurred in vivo and was important in the formation of ternary complex with JNK1. These results identify a novel protein-protein interaction between KLC1 and JLP that involves leucine zipper-like domains and support the role of motor proteins in the spatial regulation of signaling modules.

To respond properly to extracellular signals, eukaryotic cells have developed cascades of highly conserved protein kinases (MAPK family and their activating kinases), which form the central elements of signal transduction pathways that activate transcription factors in the nucleus and other effectors throughout the cell in response to environmental signals. The MAP kinase cascades have recently been shown to be regulated throughout the cell in response to environmental signals. The transcription factors in the nucleus and other effectors are the central elements of signal transduction pathways that activate transcription factors in the nucleus and other effectors throughout the cell in response to environmental signals. The MAP kinase cascades have recently been shown to be regulated throughout the cell in response to environmental signals. The transcription factors in the nucleus and other effectors are the central elements of signal transduction pathways that activate transcription factors in the nucleus and other effectors throughout the cell in response to environmental signals.
Spatial Regulation of JLP by KLC1

The direct interaction of JLP with KLC1, which is homologous to the JNK scaffolding proteins JIP-3, JSAP1, and JIP-2, 15). However, our studies also identified a novel protein-protein interaction domain within TPR that consists of a heptad repeat resembling a leucine zipper-like domain. A KLC1 protein containing point mutations within this domain hindered the ability of KLC1 to associate with JLP. Our results provide the first evidence of a leucine zipper-like domain in KLC1 that plays a role in the association between KLC and JLP. Interestingly, exogenous expression of the mutant JLP containing mutations in LZII or dominant negative form of KLC1 also resulted in the mislocalization of JLP, suggesting a role of motor proteins in the spatial regulation of signaling modules.

MATERIALS AND METHODS

Yeast Two-hybrid Screening and cDNA Cloning—The cDNA encoding the LZII of KLC1 was generated using the following primers and subcloned into an EcoRI/BamHI-digested pNLX3 yeast expression vector: sense 5'-GAGAGAATTCGAGAGATCCCTCATCACTG and antisense 5'-GAGAGAATTCGAGAGCTCAAGGGCGCCAGTCT. The construct was confirmed by sequence analysis, and the construct subsequently used to perform yeast two-hybrid screening (Fig. 1A). This approach resulted in the identification of KLC1 as a JLP interacting protein. The P19 clone was obtained from the American Type Culture Collection (Rockville, MD). Cells were cultured in Dulbecco's Modified Eagle medium supplemented with 10% fetal bovine serum (Sigma) in 6% CO2 humidified chamber. To promote cell differentiation, cells were cultured as attached monolayers on sterile Petri dishes using media supplemented with 1 μM retinoic acid (RA) for 4 days. After 4 days, the cells were harvested, washed with PBS, and then transferred to tissue culture dishes in medium lacking RA. Arabidopsis C was added to inhibit cell proliferation and to enrich the differentiated population.

Analysis of Protein-Protein Interactions in Vivo—Transfected COS-7 cells were harvested 48–72 h posttransfection and lysed in the association assay buffer (20 mM Tris-HCl (pH 7.5), 150 mm NaCl, 0.5% Nonidet P-40, 0.5 mM EDTA, 0.5 mM dithiothreitol, 2 mM Na3VO4, 1 mM phenylmethylsulfonyl fluoride, 1 mM sodium fluoride, 5 μg/ml protease inhibitors (apostatin, leupeptin, pepstatin)). Clarified lysates were rotated for 1 h at 4 °C with S-protein-agarose (Novogen) or glutathione-agarose (Sigma-Aldrich) in 2% bovine serum albumin in PBS. Coverslips were incubated with primary antibodies in PBS with 2% bovine serum albumin for 1 h. The primary antibodies used were a rabbit polyclonal antibody to JLP and a mouse monoclonal antibody to the HA epitope as described in the figure legends. Immunocomplexes were detected using Texas Red-conjugated anti-mouse or fluorescein-conjugated anti-rabbit immunoglobulin secondary antibodies (Molecular Probes) in 2% bovine serum albumin in PBS. Coverslips were then fixed with 4% paraformaldehyde in PBS for 30 min, washed with PBS, and permeabilized with 0.1% Triton X-100 in PBS. After incubation with 2% bovine serum albumin in PBS for 15 min, the coverslips were incubated with primary antibodies in PBS with 2% bovine serum albumin for 1 h. The primary antibodies used were a rabbit polyclonal antibody to JLP and a mouse monoclonal antibody to the HA epitope as described in the figure legends. Immunocomplexes were detected using Texas Red-conjugated anti-mouse or fluorescein-conjugated anti-rabbit immunoglobulin secondary antibodies (Molecular Probes) in 2% bovine serum albumin in PBS. Coverslips were mounted using the ProLong Antifade Kit (Molecular Probes) and examined by confocal microscopy.

RESULTS

Identification of KLC1 as a JLP Interacting Protein—To gain an insight into the biological functions of JLP, we sought to identify potential binding partners of the LZII of JLP using a yeast two-hybrid screening (Fig. 1A). This approach resulted in...
the identification of a partial cDNA clone of KLC1 (missing 164 bp of the 5’-sequence) as a binding partner of JLP. To confirm the specificity of the interaction, the L40 yeast strain expressing this KLC1 clone was mated with the AMR70 yeast strain expressing LZII (bait), the LZII:3–4, LZI and LZII mutants, or Lamin C, which was used as a negative control. Yeast co-expressing KLC1 and each of the LZ constructs showed growth on -Leu/-Trp plates, whereas only yeast expressing KLC1 and LZII were able to grow on -Leu/-Trp/-His plates (Fig. 1B). This suggests that KLC1 associates specifically with LZII but not with LZI. Mutation of the LZII domain by replacing the third and fourth leucine residues with alanines disrupts the KLC1/ LZII interaction.

On average, six heptad repeats of leucines comprise a leucine zipper domain. However, a minimum of only four heptad repeats of leucines are required to make up a functional leucine zipper (20). LZII of JLP is unique in that it is relatively large; it contains nine heptad repeats of leucines with the exception of a heptad repeat comprising valine at the seventh position. This implies that more than one protein can potentially bind LZII of JLP at the same time. To gain an understanding of the nature of interaction between JLP and KLC1, we investigated the specificity within LZII that associates with KLC1, it is possible that LZII is important for the proper subcellular localization of JLP. Because LZII associates with KLC1, it is possible that

In Vivo Interaction of JLP and KLC1—To validate the results performed in yeast, we sought to determine the ability of full-length JLP and KLC1 to interact when co-expressed in eukaryotic cells. GST pull-down assays using lysates derived from COS-7 cells expressing JLP and KLC1 demonstrated that GST-KLC1 could precipitate JLP-WT-HA, whereas the JLP LZII-HA mutant was unable to do so (Fig. 2A). In a reciprocal experiment, wild-type S-tagged JLP, but not the mutant JLP LZII:3–4, could precipitate KLC1 in pull-down experiments using S-protein-agarose (Fig. 2B). These results demonstrated that the association between JLP and KLC1 was dependent on LZII of JLP. Additional experiments were performed using the series of S-tagged JLP LZII mutants in conjunction with HA-KLC1 in COS-7 cells. The results of these experiments showed that although wild-type JLP, mutants JLP LZII:7–8, and JLP LZII:8–9 could precipitate KLC1, mutants JLP LZII:1–2, JLP LZII:3–4 and JLP LZII:5–6 failed to do so (Fig. 2C). These in vivo association studies in COS-7 cells corroborate the results obtained from the yeast mating assays and confirm that formation of the JLP-KLC1 complex is dependent on the first six heptad repeats within LZII of JLP (Fig. 2C). To determine the role of LZII (which associates with KLC1 in the subcellular localization of JLP), we expressed HA-tagged wild-type, LZI, and LZII mutants of JLP in NIH3T3 cells. The transfected cells were fixed, and JLP was analyzed by immunofluorescence using a specific anti-HA antibody. As described previously, wild-type JLP was mainly localized throughout the cytoplasm (Fig. 2D). No significant change in subcellular localization was observed when LZI was mutated, suggesting that LZI is not involved in the subcellular localization of JLP. However, when LZII was mutated, the JLP mutant was localized to distinct patched areas within the cytoplasm (Fig. 2D), in contrast to that observed with wild-type JLP. This indicates that LZII is important for the proper subcellular localization of JLP. Because LZII associates with KLC1, it is possible that
KLC1 may be responsible for proper localization of JLP in the cytoplasm.

Identification of a Novel Leucine Zipper-like Domain in KLC1—KLC1 contains an N-terminal α-helical domain (also known as the heptad repeat domain or coiled-coil domain) that associates with the KHC stalk and a C-terminal domain that consists of six TPR motifs (Fig. 3A). The TPR motifs are involved in protein-protein interactions and represent a link between cargo and microtubule. These TPR motifs of KLC1 have been shown to associate with JIP-1, JIP-2, JIP-3/JSAP-1/Syd, and JIP-4, but the precise domain within KLC1 that contributes to the association has not yet been identified. To determine the precise domain of KLC1 that interacts with JLP, we generated several deletion mutants of KLC1 that encoded only the heptad repeat/coiled-coil domain, the TPR domain (TPR1) or the partial TPR domain (TPR2) (Fig. 3A). S-protein precipitation studies in COS-7 cells demonstrated that S-tagged JLPWT was only able to associate with full-length KLC1 and the TPR1 mutant (Fig. 3B). However, S-tagged JLPWT was not able to co-precipitate the coiled-coil domain or TPR2 mutants. This suggests that JLPWT binds to a region in KLC1 spanning from amino acids 181 to 331. Because the LZII of JLP was found to mediate the association between JLP and KLC1, we next searched for the presence of a leucine zipper-like motif within amino acid residues 181–331 of KLC1. This search revealed the presence of a peptide sequence at position 279–300 that contained four heptad repeats (Fig. 4A). Mutation of Leu-280, Leu-287, Val-294, and Leu-301 of KLC1 to alanine (HA-KLC1LZ) abolished its association with JLP (Fig. 4B). The results of this study revealed the presence of a novel “leucine zipper-like” domain in KLC1 that was essential for binding to LZII of JLP. These results also suggest that JLP and KLC1 may from heteromers via LZII of JLP and the leucine zipper-like domain of KLC1.

Association of Endogenous KLC1 and JLP—To determine whether endogenous KLC1 and JLP could associate in vivo, we performed immunoprecipitations using cell lysates derived from NIH3T3 cells treated in the presence or absence of arsenite using control or anti-KLC1 antibodies, and subjected them to immunoblot analysis using a JLP-specific antiserum. Treatment with arsenite has been shown to stimulate the activities of JNK and p38MAPK (21, 22). In addition, our studies showed that treatment of cells with arsenite results in the phosphorylation of JLP and its perinuclear localization (data not shown). The results presented in Fig. 5 showed that the anti-KLC1 antibody could specifically precipitate JLP, demonstrating that endogenous KLC1 and JLP associate in vivo. However, treatment with arsenite did not change the levels of JLP that are associated with KLC1 (Fig. 5).

Role of LZII Domain of JLP in Subcellular Localization—The spatial organization of several cytoplasmic proteins has been shown to be dependent on their direct or indirect association with components of the cytoskeleton. KLC1 is one component that links cargo to motor proteins, which in turn are anchored on microtubules. Because our observations show that JLP associates with KLC1, we hypothesized that KLC1 was responsible for proper subcellular localization of JLP and that a dominant negative mutant of KLC1 (TPR1) could alter its subcellular localization. To address this question, we determined the subcellular distribution of JLP in human lung fibroblasts, HFL-1 (Fig. 6). The cells were infected with viral supernatants derived from packaging cells transfected with...
retroviral vectors expressing HA-tagged wild-type KLC1 and the TPR1 and TPR2 mutants shown in Fig. 3. The infected cells were then analyzed by immunofluorescence using a specific JLP antibody and an HA antibody to detect the endogenous JLP and KLC1 proteins, respectively. The results of this study showed that JLP was mainly distributed homogenously throughout the cytoplasm (Fig. 6), which is consistent with our previously published results obtained in Swiss3T3 cells (14). Moreover, wild-type KLC1 was also similarly distributed throughout the cytoplasm, suggesting that both proteins are localized in the same region of the cell (Fig. 6). In contrast, when the TPR1 mutant of KLC1 was expressed, it was detected in both the cytoplasm and the nucleus (Fig. 6). This inappropriate localization of TPR1 resulted in the translocation of JLP to the nucleus. It is possible that TPR1 transported JLP into the nucleus because of its ability to bind JLP (Fig. 3). Similarly, when the TPR2 mutant was expressed, it was detected in both the cytoplasm and the nucleus (Fig. 6). However, in this case, the mislocalization of TPR2 did not result in JLP being localized to the nucleus. This is likely because of the fact that TPR2 does not bind to JLP (Fig. 3). Taken together, these results demonstrate that the proper subcellular localization of JLP depends on its interaction with KLC1.

**Ternary Complex Formation by JLP, JNK, and KLC1**—We have previously demonstrated that JLP can regulate JNK signaling and that this phenomenon can be attributed to the ability of JLP to associate with JNK and to function as a scaffolding protein for the JNK signaling module (14). To demonstrate that JLP, KLC1, and JNK form a complex, we expressed various combinations of HA-tagged KLC1, S-tagged JLP, and JNK1 in COS-7 cells (Fig. 7). Cell lysates derived from these cells were subjected to immunoprecipitation using a KLC1-specific antibody. Our results show that when JLP or JNK1 was expressed either individually or together in the absence of KLC1, neither protein was able to be immunoprecipitated. However, when JLP was co-expressed with KLC1, JLP could be co-immunoprecipitated using the KLC1 antibody; this result is in agreement with those shown in Fig. 2. Furthermore, when KLC1 and JNK1 were co-expressed, a small
amount of JNK1 could also be co-immunoprecipitated with KLC1, which is clearly visible in the long exposure. This association was likely because of the tethering property of JLP toward KLC1 and JNK1 and is supported by the fact that greater levels of JNK1 are precipitated when exogenous JLP is co-expressed with KLC1 and JNK1 (Fig. 7). These results support the notion that JLP acts as a bridging molecule that mediates the formation of a ternary complex with KLC1 and JNK1 and rejects the notion that two independent binding events occur between JLP and KLC1, and JLP and JNK1.

**Induction of JLP and KLC1 Transcripts during Neuronal Differentiation**—The observation that JLP and KLC1 are highly expressed in adult brain compared with other tissues (4, 23) suggested that their expression might be induced during neuronal differentiation. To test this hypothesis, we examined the expression of JLP and KLC1 in the P19 cell model of neuronal differentiation in response to RA (Fig. 8A). A Northern blot analysis showed that both JLP and KLC1 were detected at low levels in proliferating P19 cells. However, when these cells were induced to differentiate, RNA levels of both JLP and KLC1 were increased as a function of time (Fig. 8B). Time course analysis indicated that JLP and KLC1 expression was not an early event following RA treatment. However, JLP and KLC1 expression was robust at day four following initiation of differentiation (Fig. 8B). The expression profiles of JLP and KLC1 are also similar at the protein level (Fig. 8C), and it is therefore likely that these proteins associate in neuronal cells as well.

**DISCUSSION**

There are essentially two classes of scaffolding proteins that assemble the JNK signaling module. The first class is comprised of the SH3-motif containing proteins known as JIP-1 and JIP-2 and the second class includes JIP-3/JSAP-1/Syd, JLP, and JIP-4, which contain leucine zipper motifs (4, 24–27). The association with KLC1 TPRs shows that it exhibits 69% homology to JIP-3/JSAP-1/Syd, with an exceptionally high homology in the two coiled-coil domains (leucine zipper domains) and Domain C. Therefore, it was not surprising that LZII of JLP was shown to associate with KLC1 when it was used as a bait in the yeast two-hybrid screening. This result is consistent with the finding that the GST-KLC1 was only able to pull down the recombinant N terminus of Syd.
Spatial Regulation of JLP by KLC1

and not the C terminus (1). Furthermore, the alternative splicing variant of JLP, JIP-4, interacts with the TPR of KLC1 via a region containing the leucine zipper (4). Our results are therefore consistent with the idea that the domains of JIP proteins that interact with KLC1 are distinct and that the binding of each JIP protein to kinesin is mediated by the TPR region of kinesin light chain.

A leucine zipper consists of a stretch of amino acids with a leucine residue in every seventh position in a coiled-coil α-helical structure (20). These leucine residues are important for the leucine zipper to form a dimer with the leucine zipper of another polypeptide. Our results provide definitive evidence that the heptad repeats of leucine residues within LZII of JLP are required for the association with KLC1 both in yeast and in mammalian cells (Figs. 1 and 2). However, to date, there have been no prior reports that identified leucine zipper-like domains in the TPRs of KLC1. We have scanned the peptide sequence of the KLC1 TPRs and identified such a domain. One stretch of amino acids contained four heptad repeats of leucine residues, with the exception of a valine in the third heptad. A KLC1 construct containing the mutations L280A, L287A, V294A, and L301A was unable to associate with JLP in mammalian cells (Fig. 4). These experiments not only show an association between JLP and KLC1 but also provide evidence for a novel leucine zipper-like domain in KLC1 that is important for the association with JLP.

The connection between motor proteins and signaling pathways has many implications. It has been reported that KLC1 is the link between kinesin motor proteins and JIPs and that the significance of this complex is to mediate proper cellular localization (26). Furthermore, the formation of a ternary complex consisting of JLP, KLC1, and JNK1 (Fig. 7) suggests that JLP serves as a link between the kinesin motor proteins and their cargo JNK signaling complex proteins (Fig. 9). Interestingly, accumulation of JLP in the perinuclear region results when cells are exposed to stress-inducing agents such as arsenite or UV radiation (14). This may represent a mechanism by which JLP can transduce signals from the periphery of the cell to the nucleus. Alternatively, scaffolding proteins could allow signaling pathways to regulate motor activity. For example, the association of kinesin with the JIPs could enable the JNK pathway to induce phosphorylation of kinesin itself or an associated protein, thereby activating the motor at the point of departure and/or inactivating it at the point of destination (Fig. 9).

We and others have demonstrated that a dominant negative mutant of kinesin (DN kinesin) can alter the proper localization of JIPs and JLP. However, it is still unresolved as to whether DN kinesin can alter the localization of JNK. In addition, it would be interesting to determine whether DN kinesin can alter the activation of JNK. It has been speculated that there is a connection between kinesin, JNK signaling, and neurogenesis. For example mice lacking the ubiquitously expressed conventional kinesin gene kif5b and mice lacking the jnk1 and jnk2 genes exhibit embryonic lethality with severe defects in early brain development (29).

Although biochemical studies and transfection assays have demonstrated that putative JNK scaffold proteins can regulate JNK activation in cultured cells, the function of these scaffold proteins in vivo has not been extensively established. One such in vivo study involves genetic alterations in the syd gene, the Drosophila homologue of JIP-3, and JASP. Mutations in syd confer axonal transport processes and resulted in massive accumulation of anterograde and retrograde membranous axonal cargo within the axons of the larval segmental nerves. Thus, proper Syd function is required for efficient axonal transport in Drosophila, and the Syd mutant phenotypes are nearly indistinguishable from mutants lacking the anterograde axonal transport motor kinesin-1 (1, 30–32). These studies suggest an important role for scaffolding proteins such as JIP-3, JASP-1, and JLP in motor transport.

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