We have isolated a 27-kDa protein that binds to cytoplasmic dynein. Microsequencing of a 17-amino acid peptide of this polypeptide yielded a sequence which completely matched the predicted sequence of the β subunit of casein kinase II, a highly conserved serine/threonine kinase. Affinity chromatography using a dynein column indicates that both the α and β subunits of casein kinase II are retained by the column from rat brain cytosol. Although dynactin is also bound to the column, casein kinase II is not a dynactin subunit. Casein kinase II does not co-immunoprecipitate with dynactin, and it binds to a dynein intermediate chain column which has been preblocked with excess p150Glued, a treatment that inhibits the binding of dynactin from cytosol. Bacterially expressed and purified rat dynein intermediate chain can be phosphorylated by casein kinase II in vitro. Further, native cytoplasmic dynein purified from rat brain can also be phosphorylated by casein kinase II in vitro. We propose that CKII may be involved in the regulation of dynein function possibly by altering its cargo specificity or its ability to interact with dynactin.

Cytoplasmic dynein is a mechanochemical ATPase that powers retrograde organelle transport in a wide variety of cell types and has been implicated in many different cellular functions, including the endocytic pathway, Golgi positioning, chromosome movement, spindle formation, nuclear positioning, and retrograde axonal transport (recently reviewed in Ref. 1). Both the intracellular targeting and the motor activity of cytoplasmic dynein are likely to be tightly regulated within the cell (reviewed in Ref. 2); however, little is as yet known about the mechanism of this regulation.

Cytoplasmic dynein is a macromolecular complex of two heavy chains (~540 kDa), approximately three intermediate chains (~74 kDa), and four electropheretically distinct light chains (~53–59 kDa). The cytoplasmic dynein intermediate chains (DICs) have been suggested to be functionally analogous to the axonemal dynein intermediate chains in targeting the dynein motor to its cargo (3). The dynein intermediate chains have also been shown to link the motor enzyme to dynactin by direct interaction with the p150Glued component of dynactin (4, 5). Dynactin is a complex oligomer of ~20 S that has been reported to increase the frequency of vesicle movement mediated by dynein in vitro (6), and which may provide an essential link between the motor and the organelle (7). Regulation of the dynein-dynactin interaction may be a key step in the mechanism of cytoplasmic dynein-mediated organelle transport.

Casein kinase II (CKII) is a ubiquitous eukaryotic serine/threonine kinase which is found in higher eukaryotes as either an αβ2 or ααββ2 heterotetramer where the α subunits (molecular mass, α = 42–44 kDa; α’ = 38 kDa) are catalytic and the β subunits (molecular mass = 26 kDa) are regulatory (for reviews, see Refs. 8–11). Substrates of CKII include cytoskeletal proteins such as myosin heavy and light chains, tropomyosin-T, β-tubulin, tau, and MAP-1B (8). CKII function has been implicated in processes of cell cycle regulation, cell division, and signal transduction (12, 13). Although the majority of cellular CKII localizes to the nucleus, antibodies directed against CKII also localize to the centrosome (14). Immunolocalization studies indicate that a population of cellular CKII is associated with the spindle apparatus in dividing cells (14, 15). Microinjection studies have also suggested a role for CKII in the regulation of neurite outgrowth, as the microinjection of antisense oligonucleotides to CKIIα blocked neuritogenesis (16). These studies suggest that CKII may play a role both in regulating the cell cycle and in neurite extension; cytoplasmic dynein is thought to be involved in each of these processes.

In this study we isolated CKII as a cytoplasmic dynein-binding protein using affinity chromatography (5). We demonstrate that CKII binds to the intermediate chain of dynein, but is not a component of the dynactin complex, and that CKII is capable of phosphorylating the dynein intermediate chain in vitro. This phosphorylation of DIC by CKII may modulate dynein function at the level of dynein complex assembly, motor activity, or specificity of interaction with its cargo, and potentially may play a role in modulating the interphase and mitotic functions of cytoplasmic dynein.

MATERIALS AND METHODS

Preparation of Cytosol and Purification of Proteins—Rat brain cytosol was prepared as described previously (5) and either loaded onto a dynein affinity column or used for cytoplasmic dynein preparation by microtubule affinity (34). In some experiments purified cytoplasmic dynein was treated with lambda protein phosphatase (New England Biolabs, Beverly, MA); the phosphatase was removed by linear sucrose density gradient centrifugation. Recombinant cytoplasmic dynein intermediate chain was expressed in Escherichia coli and purified as described previously (5).

Affinity Chromatography—Dynein intermediate chain cross-linked to CH-Sepharose 4B beads at a concentration of ~2 mg ligand/ml of drained beads was used for all affinity chromatography experiments. For isolation of novel dynein-binding proteins, 10 ml of brain cytosol was loaded onto a 2-ml dynein affinity column and the column was washed with 50 volumes of PHEM (50 mM Na-HEPES, 50 mM Na-
PIpes, 1 mM EDTA, 1 mM MgCl₂, pH 6.9) buffer. This extensive wash was followed by a 2-bed volume wash with 25 mM NaCl in PHEM. The column was then eluted with PHEM containing 1 M NaCl and dialyzed against PHEM containing 0.2 mM diithiothreitol, then resolved on a 5–20% sucrose gradient. The resulting fractions were analyzed by SDSPAGE, and those fractions near the 20 S peak were pooled and methanol-precipitated. For other experiments, the column size was reduced to 0.5-mL bed volumes and processed as described above and eluted with 1 M NaCl in PHEM. The eluate fractions were methanol-precipitated. The precipitates were resolved by 10% SDS-PAGE, transferred onto Immobilon-P (Millipore, Bedford, MA), and probed with anti-human CKII, rabbit polyclonal antibody (rabbit polyclonal, Upstate Biotechnology, Inc., Lake Placid, NY) or anti-CKIIβ chain antibody (mouse monoclonal, Transduction Laboratories, Lexington, KY).

Immunoprecipitation—Immunoprecipitation from rat brain cytosol was carried out as described previously (17) with an affinity-purified rabbit polyclonal anti-p150000 chain antibody. The immunoprecipitate was then resolved by SDS-PAGE, transferred to Immobilon-P, and probed with anti-p150000, anti-p50, anti-centrin, and anti-CKIIβ antibodies.

Peptide Sequencing—The methanol precipitate of the eluate from the dynein affinity column was resolved by 10% SDS-PAGE, transferred to Immobilon-P, and briefly stained with Ponceau S. A polypeptide band of 27 kDa was excised, subjected to tryptic digest, and subsequently microsequenced.

In Vitro Phosphorylation—Recombinant dynein intermediate chain was equilibrated in PHEM buffer using a NAP-10 gel filtration column (Pharmacia, Uppsala, Sweden). In six identical tubes 10 μg of DIC, 250 μM Mg-ATP, 10 μCi of [γ-32P]ATP (DuPont NEN), 1000 units of CKII (human recombinant, New England Biolabs), and 1 × CKII buffer were mixed, and the final volume brought to 20 μL. An additional tube containing no CKII was also included to serve as a control. Reactions were terminated by addition of 5 × SDS sample buffer and boiling. For in vitro phosphorylation of native cytoplasmic dynein 10 μg of protein (λ-DIC) was treated (or untreated) was mixed with 250 μM Mg-ATP, 40 μCi of [γ-32P]ATP, and 1 × CKII buffer and reacted at 30 °C. The phosphorylated samples were resolved on 8% or 10% SDS-PAGE as noted, and the dried gels were exposed to x-ray film. The extent of phosphate incorporation was calculated by excising the gel bands and counting the radioactivity by using a scintillation counter. Protein concentration in the bands was determined by densitometric analysis of the Coomassie-stained gel, using BSA as a standard.

RESULTS

Casein Kinase II Binds to Dynein in Vitro—We have previously demonstrated that a dynein intermediate chain affinity column retains dynactin complex from brain cytosol (5). We extended this technique to identify other potential dynein-binding proteins. Rat brain cytosol was loaded onto an intermediate chain affinity column and after extensive washing, the column was eluted with 1 M NaCl. Initially, we resolved the eluate on a sucrose gradient and the fractions corresponding to −20 S were pooled and methanol precipitated. Analysis of the precipitate by SDSPAGE revealed an uncharacterized polypeptide of 27 kDa (Fig. 1A). Amino-terminal amino acid microsequencing of the resulting peptides on a 477A Sequenator (Applied Biosystems, Foster City, CA).

We have previously shown that casein kinase II (CKII), cytosol-loaded; lanes 2–4, DIC eluate was concentrated and subsequently probed with antibodies to CKIIα and CKIIβ (B). Lane 1, cytosol-loaded; lanes 2 and 5, flow-throughs; lanes 3 and 6, final wash; lanes 4 and 7, elution with 1 M NaCl.

FIG. 1. Polypeptides retained by a dynein affinity column and the comparison of a peptide sequence from p27 with the predicted sequence of rat CKIIβ. A, DIC eluate was concentrated and loaded on a 5–20% sucrose gradient. Fractions corresponding to –20 S were pooled, methanol precipitated, resolved by SDS-PAGE, transferred to Immobilon, and Coomassie-stained. This gel lane shows all the reported subunits of dynactin. The band corresponding to 27 kDa was excised, subjected to tryptic digest, and subsequently microsequenced. B, the upper line shows the sequence of the peptide from p27, and the lower line shows matching sequence from rat CKIIβ (19). The arrows indicate the predicted cleavage sites for trypsin.

Since CKII exists as a tetramer of 2α chains and 2β chains in all higher eukaryotes, we verified that both α and β chains are retained on a dynein affinity column as shown in Fig. 2. Rat brain cytosol was loaded on a dynein column, which was then washed extensively and eluted with 1 M NaCl. The resulting fractions were resolved by SDS-PAGE, transferred onto Immobilon-P, Coomassie-stained for total protein (Fig. 2A) and subsequently probed for CKII α and β chain using rabbit polyclonal and mouse monoclonal antibodies specific for these polypeptides, respectively (Fig. 2B). As shown in Fig. 2, the immunoblot indicated that the cytosol contains CKIIα (lane 1), which when loaded on to the dynein affinity column (lanes 2–4) binds to dynein. Not all CKII binds to dynein as indicated by the presence of CKIIα in the flow-through samples (lanes 2 and 5); this may suggest that the column capacity is exceeded or that there is a pool of cytosolic CKII that does not bind to dynein. The 1 M NaCl eluate of the dynein column (lane 4) contains both α and β chains of CKII suggesting that intact CKII holoenzyme binds to the dynein column. A BSA control column which was constructed identically did not retain any CKII from cytosol.
Casein Kinase II Binds to Cytoplasmic Dynein

While there was no detectable β chain in the cytosol as compared to a chain, the β chain is clearly visible in the salt eluate of the dynein column suggesting that the eluate fraction is highly enriched in CKII. Difficulty in detecting the β subunit of CKII in the total cytosol may be due to weaker immunoreactivity of the β antibody. Alternatively, there may be a lower level of β subunits present in the cytosol (Fig. 2B, lane 1). Some studies support the hypothesis that there is a limited pool of CKβ relative to α subunits (35), and in some species, β subunits are completely lacking (36, 37).

CKII Is Not a Dynactin Subunit—It has been reported that there is a 27-kDa dynactin polypeptide (20). To test the possibility that the 27-kDa CKII β polypeptide might be a subunit of dynactin, we performed immunoprecipitation using an affinity-purified anti-p150\(^{Glued}\) antibody (Fig. 3). The immunoprecipitate was observed to contain the dynactin subunits p150\(^{Glued}\), p50 (dynamitin), and centractin (Arp1) (lane 2), while only 2% of centractin bound to such a column pretreated with p150\(^{Glued}\). This result suggests that CKII is not the reported 27-kDa dynactin subunit.

CKII Phosphorylates DIC in Vitro—Primary amino acid sequence analysis of the rat dynein intermediate chain revealed 11 potential CKII phosphorylation sites. To investigate whether CKII could phosphorylate dynein in vitro, we incubated bacterially expressed and purified rat DIC with CKII and \(\gamma\)-P\(^{32}\)ATP. The resulting time course shown in Fig. 5A indicates that dynein is readily phosphorylated by purified CKII (lanes 2–7). Incorporation of phosphate into the dynein intermediate chain in this experiment approached 2 mol of phosphate per mol of polypeptide (Fig. 5C). This suggests that there are at least 2 CKII phosphorylation sites in DIC.

We also tested whether dynein in its native state could be phosphorylated in vitro by CKII. Purified cytoplasmic dynein was incubated with recombinant CKII at 30 °C, and the reactions were analyzed by SDS-PAGE followed by autoradiography (Fig. 5B). The intermediate chain of cytoplasmic dynein was phosphorylated, as well as the dynein heavy chain (lanes 4–6). However, the observed extent of phosphorylation was relatively low. We hypothesized that this low level of incorporation might be because the dynein was phosphorylated as isolated (21, 22). Therefore we pretreated the purified dynein with lambda protein phosphatase, removed the phosphatase by sucrose gradient centrifugation, and then compared the phosphorylation of the phosphatase-treated dynein with that of untreated dynein (lanes 1–3). Phosphorylation of the intermediate chain was increased by -2-fold (as judged by densitometric analysis) following phosphatase treatment (lane 3 versus lane 6), to a level of 0.5 mol of phosphate incorporated per mol of the intermediate chain. Taken together, these results suggest that CKII is capable of phosphorylating the intermediate chain in native dynein and that the intermediate chain in the

![Fig. 3](image-url)  
**Fig. 3.** CKII does not co-precipitate with dynactin. High speed supernatant of brain cytosol (lane 1) was immunoprecipitated with p150\(^{Glued}\) antibody (lane 3) or control Sepharose beads (lane 2), the immunoprecipitate resolved on SDS-PAGE, transferred onto Immobilon-P, Coomassie-stained (A), and subsequently probed for p150\(^{Glued}\), p50, centractin, and CKIIα (B). While all subunits of dynactin are present in both immunoprecipitate and column elution, CKII is present only in the DIC eluate. Lane 1, total cytosol used for immunoprecipitation; lane 2, control immunoprecipitation using Sepharose beads alone without antibody; lane 3, immunoprecipitate using p150\(^{Glued}\) antibody; lane 4, elution from a dynein column for comparison.

![Fig. 4](image-url)  
**Fig. 4.** CKII binds to a dynein column independently from dynactin. Rat brain cytosol (lane 1) was loaded on intermediate chain column that was either pretreated with BSA (lanes 2–4) or a p150\(^{Glued}\) fragment (lanes 5–7). The columns were washed and eluted with 1.0 M NaCl and fractions resolved by SDS-PAGE, transferred onto Immobilon-P, Coomassie-stained (A), and subsequently probed for CKIIα or centractin (B) as indicated. Results show that while dynactin was blocked by p150\(^{Glued}\) pretreatment, CKII was not. Lane 1, cytosol input; lanes 2 and 5, flow throughs; lanes 3 and 6, final wash; lanes 4 and 7, elution with 1 M NaCl.
Phosphorylation of dynein by CKII in vitro. A, bacterially expressed and purified intermediate chain was subjected to CKII phosphorylation in vitro for the following time intervals, lane 2, 5 min; lane 3, 10 min; lane 4, 15 min; lane 5, 30 min; lane 6, 60 min; lane 7, 120 min. Lane 1 is a 15-min control sample lacking CKII. The reactions were stopped by adding 5 x SDS sample buffer to the reaction tubes and boiling before analysis by SDS-PAGE followed by autoradiography. The bands corresponding to polypeptides of 27 and 24 kDa are amino-terminal fragments of the dynein intermediate chain. B, purified dynein from rat brain cytosol was subjected to in vitro phosphorylation by CKII with (lanes 1-3) or without (lanes 4-6) lambda protein phosphatase pretreatment. Control samples (lanes 7-9) did not contain any CKII. The reactions were stopped at 45 min (lanes 1, 4, and 7), 75 min (lanes 2, 5, and 8), and 120 min (lanes 3, 6, and 9). The subsequent analysis was done as described in A. The results show that dynein is phosphorylated by CKII and phosphatase treatment enhances the amount of radiolabeled ATP incorporation. C, graph depicts moles of phosphate incorporated into a mole of recombinant DIC in vitro by CKII. This experiment was done separately from A and indicates that, at saturation, approximately 2 phosphate moles are incorporated per DIC molecule.

The intermediate chains of cytoplasmic dynein have been suggested to play a role in the targeting of the motor (3). Recent biochemical evidence demonstrating direct binding between cytoplasmic dynein intermediate chains and the p150Glucl component of dynactin (4, 5) also point to a potential targeting role for the intermediate chains. Using an affinity column assay we have described previously (5), we have now determined that the α and β subunits of CKII bind to a dynein affinity column (Fig. 1) and that the first 120 amino acids of the DIC are sufficient for CKII binding (data not shown).

The size of the CKIIβ subunit (molecular mass = 26 kDa) matches closely with the reported 27-kDa subunit of the dynactin complex (20). Thus there was the possibility that CKIIβ may represent this as yet uncharacterized dynactin subunit since it is known that dynactin complex binds to the intermediate chain column. We tested this possibility by immunoprecipitation as well as in column blocking experiments. The results clearly demonstrate that CKIIβ cannot be a dynactin component since it does not co-precipitate with other dynactin subunits nor is its binding to dynein affected by pretreatment of the column with p150Glucl. The pretreatment of the dynein column with excess p150Glucl blocked subsequent binding of dynactin as evidenced by lack of retention of centractin (Arp1; Fig. 4, lane 4) on such a column. This result establishes that association of CKII with dynein is independent of the dynine-dynactin interaction. However, we have not yet determined whether the binding of CKII to dynein is direct or is indirect and mediated by an as yet unidentified factor. It also remains to be determined if there is a true 27-kDa subunit of dynactin or whether containing CKIIβ subunit was taken to be a dynactin subunit (20). Using affinity chromatography and immunoprecipitation assays, we have recently demonstrated that actin is not a stoichiometric component of dynactin (7), as was suggested in an earlier report (20).

The observation that CKII binds to dynein is of particular interest since CKII has been reported to interact with a number of proteins, some of which are thought to be physiological substrates of CKII (23–31). For example, nucleoplasmin, a nuclear protein, associates with and is phosphorylated by CKII (24). Similarly, FK506-binding protein (a 25-kDa nuclear protein) also associates with CKII (31). Finally, CKII copurifies with proteosomes and phosphorylates the 30-kDa proteosome subunit (23). Since the CKII heterotetramer bound to dynein, we tested whether CKII could phosphorylate dynein in vitro. The results presented here demonstrate that bacterially expressed dynein intermediate chain is phosphorylated by recombinant CKII. We also show that intermediate chains in the purified dynein complex can also be phosphorylated by CKII.

The observation that pretreatment of cytoplasmic dynein with a phosphatase increases the subsequent phosphorylation by CKII (Fig. 4) lends support to the hypothesis that intermediate chains in the dynein complex are phosphorylated within the cell, potentially on CKII-specific sites.

Despite cytoplasmic dynein’s potential roles in a wide range of cellular events, very little is known about how a single major form of cytoplasmic dynein can participate in a regulated manner in all of these processes. One key regulatory mechanism for cytoplasmic dynein may be its phosphorylation (see Ref. 2 for a recent review). CKII is an ubiquitous serine/threonine kinase with an established role in cell proliferation. While the CKII regulatory pathway is not well understood, it is interesting to note that CKII has been shown to localize to the spindle apparatus and centrosome; cytoplasmic dynein has also been shown to localize to these cellular structures (1).

Dynein phosphorylation may affect the affinity of the enzyme for its cellular cargo. Several studies have noted a correlation between dynein phosphorylation and its distribution on membranous organelles (22, 32, 33). Phosphorylation may also affect the mechanochemical cycle of cytoplasmic dynein. It has been reported that dynein undergoing anterograde transport (therefore in the inactive state) is differentially phosphorylated than the general dynein pool (21), suggesting that the phosphorylation of dynein may regulate its motor activity. It is possible that other polypeptides either in dynein or in the closely associated dynactin complex are physiological substrates of CKII. As described above, we have noted the phosphorylation of dynein heavy chain in vitro by CKII; our prelimi-
inary observations also suggest that some of the dynactin polypeptides are targets for phosphorylation by this kinase.

In summary, CKII alone or in conjunction with other kinases may modify DIC in such a way as to modulate dynein function at the level of complex assembly, dynein activity (motor or ATPase), or cargo specificity. The association of a kinase with dynein which we have observed may allow for modification of not only the intermediate chains of dynein but also the heavy and light chains, as well as other accessory proteins such as dynactin. While the functional implications of this CKII-dynein association remain to be explored, the observation that CKII can bind to and phosphorylate cytoplasmic dynein should be a significant step toward understanding the regulation of dynein function within the cell.

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