A novel murine membrane-associated protein kinase, PKK (protein kinase C-associated kinase), was cloned on the basis of its physical association with protein kinase Cβ (PKCβ). The regulated expression of PKK in mouse embryos is consistent with a role for this kinase in early embryogenesis. The human homolog of PKK has over 90% identity to its murine counterpart, has been localized to chromosome 21q22.3, and is identical to the PKCβ-interacting kinase, DIK (Bahr, C., Rohwer, A., Stemple, L., Rincke, G., Marks, F., and Gschwendt, M. (2000) J. Biol. Chem. 275, 36350–36357). PKK comprises an N-terminal kinase domain and a C-terminal region containing 11 ankyrin repeats. PKK exhibits protein kinase activity in vitro and associates with cellular membranes. PKK exists in three discernible forms at steady state: an underphosphorylated form of 100 kDa; a soluble, cytosolic, phosphorylated form of 110 kDa; and a phosphorylated, detergent-insoluble form of 112 kDa. PKK is initially synthesized as an underphosphorylated soluble 100-kDa protein that is quantitatively converted to a detergent-soluble 110-kDa form. This conversion requires an active catalytic domain. Although PKK physically associates with PKCβ, it does not phosphorylate this PKC isoform. However, PKK itself may be phosphorylated by PKCβ. PKK represents a developmentally regulated protein kinase that can associate with membranes. The functional significance of its association with PKCβ remains to be ascertained.

The protein kinase C (PKC) family is made up of at least 11 phospholipid-dependent serine/threonine kinases (1–4). PKC was originally described as a calcium-activated, phospholipid-dependent serine/threonine kinase. Conventional PKCs include PKCα, PKCβⅠ, PKCβⅡ, and PKCγ. They require phosophidylerine, diacylglycerol, and Ca2+ for maximal activation. PKC subfamilies exist which either do not require calcium (novel PKCs) or which require neither calcium nor diacylglycerol for optimal activation (atypical PKCs). Specific PKC isozymes play important and distinct roles in the development and activation of most vertebrate cell types including lymphocytes, but the study of the function of individual isozymes is in its infancy.

PKCβⅠ and PKCβⅡ are generated by alternative splicing at the PKCβ locus and differ over a 50–52-amino acid stretch at their C termini. This locus has been shown to be critical for the generation of B-1 B lymphocytes and for the activation of peripheral B cells by T-independent antigens (5). PKCβ has also been shown to be required in HL-60 cells for phorbol 12-myristate 13-acetate-induced differentiation into macrophages (6, 7). One of the best studied substrates of PKCs in general, MARCKS (myristoylated alanine rich protein kinase C substrate), is also a substrate of PKCβ (9). A number of other proteins shown to be phosphorylated by PKCβ and/or PKCβII include LIM domain proteins (10), Cut homeodomain proteins (11), tyrosinase (12), nuclear lamins (13), PKC-interacting protein-1 (14), and F-actin (15). Although a number of proteins that are phosphorylated by PKCβ have been described, the substrate(s) of PKCβ critical for B cell development remains to be identified.

We describe here a novel protein kinase that can physically associate with PKCβ. We show that this novel ankyrin repeat-containing kinase, which we call PKK (for PKC-associated kinase), is associated with membranes, is initially synthesized as a soluble 100-kDa protein, and is subsequently converted to a larger phosphorylated form. This conversion depends, at least in part, on the intrinsic catalytic activity of this kinase. At steady state, a portion of this protein is membrane-associated. The significance of the association of PKK with PKCβ remains to be determined.

**EXPERIMENTAL PROCEDURES**

Plasmids—HA-tagged PKCβⅠ, PKCα, and PKCβ plasmids were generous gifts from Dr. Shun’ichi Kuroda (10), Dr. Peter Parker (16), and Dr. Weiqun Li (17), respectively. The kinase domains of PKCβ (residues 291–673), PKCβⅠ (residues 291–673), and PKCα (residues 343–674) were used as baits in a yeast two-hybrid approach and were individually cloned into the SalI/SpeI sites of pPC97AD (Life Technologies, Inc.; kindly provided by Dr. Marc Vidal, Dana-Farber Cancer Institute). A kinase dead PKK K51R mutant, was created using polymerase chain reaction-based mutagenesis and confirmed by sequencing. In-frame glutathione S-transferase (GST) fusion constructs involving distinct fragments of PKK were generated using pGEX2T (Amersham Pharmacia Biotech).

A Yeast Two-hybrid Assay for Proteins That Interact with the Catalytic Domain of PKCβ—I A yeast two-hybrid screen was performed as described (18). A pC86DB-cDNA library was made with 12–13-day whole murine fetuses (gift of Dr. Joshua LaBaer, Harvard Medical School). The expression of bait plasmids was confirmed by Western
blotting using individual isotype-specific antibodies. For library screening, the yeast MaV103 strain carrying the pPC97AD-PKC\textsuperscript{b}1-(291–673) plasmid was transformed with the pPC86DB cDNA library. Transforms were replica-plated onto different selective plates as described (18). The specificity of cDNAs obtained from positive yeast colonies was analyzed by retransformation with different controls. Both strands of a 3.4-kilobase pair full-length cDNA clone obtained from the two-hybrid assay were sequenced, and this insert was cloned into a pCMV-driven vector.

**a**. The 10 potential PKC phosphorylation sites are underlined. The GenBank accession number is AF302127.

**b**. Alignment of peptide sequences of mouse and human PKK. A sequence comparison was carried out using the GeneWorks program. Identical residues are boxed.

**c**. A schematic representation of PKK.

**Fig. 1.** PKK is a novel ankyrin-repeat-containing kinase.
expression vector with a Flag tag.

In Situ Hybridization—Whole mount in situ hybridization on 8.5–10.5-day post coitum (dpc) WB mouse embryos was carried out as described (19). In situ hybridization on frozen sections of paraformaldehyde-fixed mouse embryonic tissues with digoxigenin-labeled antisense probes was carried out essentially as described by Ma et al. (20). Photomicrographs of whole mount embryos or sections following in situ hybridization or immunolabeling were collected on an Olympus SZH10 stereo dissecting microscope or Nikon E600 compound microscope fitted with a SPOT I digital camera (Diagnostic Imaging). Digoxigenin-labeled PKK, PKCβI, and PKCβII probes were used in these studies.

Transfections, Immunoprecipitation, and Kinase Assays—293T cells were transiently transfected with different plasmid constructs using the calcium phosphate precipitation method as described (21). Harvested cells were washed once with cold PBS and lysed in lysis buffer (1 ml of 50 mM Tris, pH 7.5, 150 mM NaCl, 1 mM EDTA, 10 mM β-mercaptoethanol, 50 mM NaF, 1 mM Na3VO4, 1 mM phenylmethysulfonyl fluoride, 10 μg pepstatin, and 1% (v/v) Triton X-100). Nuclei were removed by centrifugation, and the cell lysates were incubated for 30 min on ice with either 3 μg of anti-Flag (M2, Eastman Kodak Co.) or anti-HA (12CA5, Roche Molecular Biochemicals) monoclonal antibody followed by incubation with 10 μl of protein G-Sepharose 4 fast flow (50% slurry, Amersham Pharmacia Biotech) at 4 °C for 1 h. The beads were washed four times with lysis buffer and twice with kinase buffer (see below). Half of the beads were reserved for Western blotting. The other half of each sample was used in an in vitro kinase assay. Beads were sus-
The expression of PKK is developmentally regulated. A Northern blot analysis of PKK mRNA. A blot of polyadenylated mRNA isolated from various adult mouse tissues (CLONTECH) was used. Only a single 4.4-kilobase transcript of PKK was detected (upper panel). The blot was reprobed with mouse β-actin (lower panel). b—g, analysis of PKK, PKCβII, PKCβIII expression in the developing mouse embryos. b—d, mouse embryos at 10.5 dpc were subjected to whole mount in situ hybridization. b, b—d, early expression of PKK in the developing telencephalon (t), diencephalon (d), otic vesicle (ot), branchial arches, gut (gt), and genitourinary systems. The closed arrow indicates expression in the primitive vessels, and the open arrow shows expression in the branchial arches. c and d, expression of PKCβII (c) and PKCβIII (d) overlapped that of PKK in the primitive vessels (closed arrow) and the branch arch primordium (open arrow). Note that a scattered population of PKC-expressing cells was present in the spinal cord. e, at 12.5 dpc, ongoing expression in the gut (gt) and genitourinary system (e.g. genital tubercle (gt)) is detected, as well as transient expression in the ventral neural tube (arrow). f and g, at 14.5 dpc, strong expression throughout the gastrointestinal tract is detected (es, esophagus; du, duodenum; mg, mid gut). The arrow in panel f indicates expression in the skin.

RESULTS

Identification of a Protein That Interacts with PKCβ—A two-hybrid screen was initiated using the catalytic domain of PKCβII as a bait. Our initial goal was either to identify targets of PKCβ or to identify proteins that might regulate the activity of PKCβ. In particular we were aware that we might identify protein kinases, other than PDK1 (22), required for the activation of PKCβ. The yeast strain used in this two-hybrid assay...
contains three stably integrated reporter constructs expressing HIS3, LacZ, and URA3. Clones were considered to have scored positively if they grew on plates lacking histidine and containing 3-AT, if they turned blue on 5-bromo-4-chloro-3-indolyl β-d-galactopyranoside (X-gal) plates, and if they grew on plates lacking uracil but failed to grow in 5-fluoroorotic acid (5-FOA), which is toxic if URA3 is expressed. 4 × 10⁶ independent transformants from a mouse embryo library were screened using this stringent approach. Only one positive clone was obtained. Rescreening with specific baits revealed that the product of this clone interacted specifically with the catalytic domain of PKCβI but not with a number of other baits including the catalytic domain of PKCβII, the Rb-related p130 pocket protein, the cytoplasmic tail of CD44, or the DP1 subunit of E2F-DP complexes.

**PKK Is a Novel Ankyrin Repeat-containing Protein Kinase**—We obtained a full-length clone from a murine embryonic cDNA library; the sequence of this cDNA is depicted in Fig. 1a. The PKK cDNA contains a 2358-base pair open reading frame with a presumptive initiator methionine at nucleotide 50, a TAG stop codon at nucleotide 2408, and an AUAATA polyadenylation signal at nucleotide 3527, 11 base pairs upstream of the cleavage site. An in-frame stop codon was identified upstream of the first ATG codon. Preliminary analysis of the predicted protein sequence revealed a novel putative serine/threonine kinase with the kinase domain encompassing approximately the first 300 amino acids. A stretch of 11 ankyrin repeats was noted in the C-terminal half of the open reading frame starting at residue 442 (schematically represented in Fig. 1c). In *in vitro* transcription and translation in a rabbit reticulocyte lysate yielded a protein with an apparent molecular mass of 100 kDa (data not shown). Although no close homologs of this cDNA were noted when this manuscript was originally prepared for submission (see “Discussion”), an analysis of the 12 conserved kinase subdomains suggested that it encoded a novel protein kinase. The predicted protein encoded by this cDNA was called PKK (for PKC-associated kinase). An examination of subdomains VI and VII suggested that PKK represents a serine/threonine kinase with conservation of a DLKPAN motif and a DFG triplet (29), but the possibility that it represents a dual specificity serine/threonine and tyrosine kinase was not excluded. The activation loop of PKK contains a Ser-X-X-Ser motif characteristically found in MAP kinase kinases (24), which are typically dual specificity kinases, and in related kinases such as I KK1 and I KK2 (25). Ten potential PKK phosphorylation sites, were noted and these are underlined in Fig. 1a. A human PKK cDNA was obtained using the mouse insert as a probe. Polymerase chain reaction analysis of human/rodent radiation hybrids localized PKK to human chromosome 21. The sequence of human chromosome 21 was published while this manuscript was in preparation (26), and we confirmed the localization of human PKK to chromosome 21q22.3. The complete sequence of human PKK was confirmed from the human chromosome 21 sequence data base (26). The mouse and human cDNAs encode almost identical proteins, but the mouse sequence includes two residues (Glu¹¹⁸ and Ser¹¹⁷) in the linker region between the kinase domain and the ankyrin repeats that were not predicted by the human sequence (Fig. 1b).

Northern blots (Fig. 2a and data not shown) revealed that PKK is widely expressed in most tissues but is expressed at very low levels in the spleen. PKK is, however, relatively abundant in the thymus and is expressed in bone marrow in pre-B, pre-B, and immature B cells (data not shown).

During embryogenesis, PKK mRNA transcripts were detected at 10.5 dpc at diverse locations including the embryonic forebrain, otic vesicle, branchial arches, primitive gut, and genitourinary system (Fig. 2b). Expression of PKCβI and PKCβII (Fig. 2, c and d) broadly mimicked that of PKK in the primitive vessels and branchial arch primordium, although PKK and PKCβ expression only partially overlap during development. At 12.5 dpc, ongoing expression was detected in the gut, the mesonephric duct of the genitourinary tract, and the urogenital sinus. Interestingly, we noted transient expression of PKK in the ventral neural tube at 12.5 dpc (arrow, Fig. 2e) but not at the earlier or later stages tested. By 14.5 dpc, strong expression throughout the gastrointestinal tract was observed in the luminal tissues of the esophagus, stomach, duodenum, and intestines (Fig. 2, f and g), as well as transient expression in the skin. In addition, PKK was expressed in the collecting system of the genitourinary tract but not in kidney. These results are consistent with the possibility that PKK has roles during embryonic development.

In Fig. 3, we provide formal evidence that PKK is a protein kinase. In an immunoprecipitation kinase assay, autophosphorylation of PKK was observed, and this kinase also phosphorylates exogenous substrates such as myelin basic protein (data not shown) and histone H1 (Fig. 3). These findings do not rule out the possibility that an associated kinase (such as PKCβ) may mediate some of these activities. However, the PKK K51R mutant (kinase dead PKK) has limited immunoprecipitation kinase activity, indicating that PKK is indeed a kinase (Fig. 3). PKK is frequently visualized in transfected cells as a doublet in the 100–110 kDa range (see Figs. 4-7). The relationship between some of the different forms of this protein has been established and is described below (see Figs. 6 and 7).

**PKK and PKCβ Can Physically Associate with One Another**—We have confirmed that PKK and PKCβ can associate with each

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**Fig. 4. PKK interacts with PKCβI.** HA-tagged PKCβI was co-transfected with vector alone or with Flag-tagged wild type PKK or PKK K51R into 293T cells. Cell lysates were immunoprecipitated either with an anti-Flag antibody or with anti-HA antibody. The immunoprecipitates (IP) were analyzed by immunoblotting (WB; Western blot) with anti-HA or anti-Flag antibodies. An aliquot (one-tenth of the total cell lysate) was used for immunoblotting with the corresponding anti-tag antibodies as indicated.
terminal portions of PKK (residues 402–786 and 461–786) were phosphorylated in vitro, whereas a fusion protein including the N-terminal portion (residues 1–320) was not. However, in numerous experiments in which PKK was coexpressed with PKCβ or with PKCδ, we were unable to demonstrate a reproducible and significant enhancement of the catalytic activity of PKK by either of these PKCs (data not shown).

Since PKK apparently exists in multiple forms, we decided to examine the subcellular localization of these distinct forms and to determine whether their generation depended on post-translational modification events.

**PKK Exists in Multiple Cytosolic and Membrane-associated Forms**—When COS and 293T cells transfected with Flag-tagged PKK were permeabilized and stained using indirect immunofluorescence, a fine reticular staining pattern was observed, suggesting that PKK associates with intracellular membranes (Fig. 6a). It was clear, however, that PKK was not present in the nucleus. Examination of PKK by subcellular fractionation (Fig. 6b) revealed that a major portion of cellular PKK was cytosolic, the majority of this form being seen as a 110-kDa protein. It is possible that a proportion of this cytosolic protein is actually loosely associated with membranes in the cell, in keeping with our immunofluorescence results. Extraction of membranes with 1% Triton X-100 revealed no PKK, but two forms of PKK were noted in the Triton-insoluble membrane fraction, one of 100 kDa and another of 112 kDa (Fig. 6b). Because we were concerned that a considerable amount of PKK may have been discarded in the nuclear fraction, we examined the Triton X-100-extracted supernatants and pellets of transfected 293T cells (Fig. 6c, left panel). We also performed subcellular fractionation studies in which we separated the cytosol from all pooled membrane fractions in the cell including the membranes of the nuclear envelope. These separations were performed both with 293T cells transfected with wild type PKK as well as with cells transfected with K51R PKK (Fig. 6c, right panel). As depicted in Fig. 6c, the major Triton X-100-soluble form of PKK migrates at 110 kDa, and a minor amount of this underphosphorylated 100-kDa form was also seen in the Triton X-100 supernatant. The soluble 110-kDa form corresponds to the 110-kDa form seen in the cytosol (Fig. 6, b and c, right panel). When detergent-resistant cell pellets were solubilized in SDS loading buffer, a 112-kDa form and a 100-kDa form of PKK were observed. Both the Triton X-100-resistant 112-kDa form and the 110-kDa Triton X-100-extractable form of PKK yield a 100-kDa species when subjected to lambda phosphatase treatment (Fig. 6d). There are therefore three major forms of PKK that can be identified. Most of the PKK in the cell is a soluble, primarily cytosolic or loosely membrane-associated, phosphorylated 110-kDa form. A 100-kDa under- or nonphosphorylated form of PKK represents a small proportion of cytosolic PKK but makes up about one-half of the Triton X-100-insoluble fraction of PKK. A 112-kDa hyperphosphorylated detergent-insoluble form of PKK was also readily identified. It is presumed that this protein is associated with cellular membranes or with cytoskeletal elements. Although it is possible that a portion of PKK may exist in detergent-insoluble glycolipid domains or lipid rafts, immunofluorescence studies did not reveal discernible accumulations of PKK on the inside of the plasma membrane in transfected cells (Fig. 6a).

Conversely, PKK from an Underphosphorylated 100-kDa Species to a 110-kDa Phosphorylated Form Occurs Post-synthetically and Requires the Catalytic Activity of PKK—In tran-
siently transfected cells pulse-labeled with \[^{35}\text{S}\]methionine for 10 min, wild-type PKK is initially synthesized as a 100-kDa protein. In cells that have been chased in complete medium for 20 min or longer, this 100-kDa protein is quantitatively converted into a larger 110-kDa form. Experiments in which PKK was examined after a 40-min or 1-h chase period are depicted in Fig. 7a, left panel, and b, respectively. A similar conversion to the larger form has been noted after 20 min of chase, but only the 100-kDa form is seen after a 10-min chase period (data not shown). The post-synthetically derived larger form of PKK can be reduced to a 100-kDa form by treatment with lambda phosphatase, confirming that it is derived by phosphorylation (Fig. in which equivalent amounts of phosphatase-treated and untreated pellets were separated is depicted in the right panel. PKK was revealed by an anti-Flag Western blot.

**Fig. 6.** PKK exists in multiple underphosphorylated and phosphorylated forms. a, COS cells were transfected with Flag-tagged wild type PKK, and permeabilized cells were stained with anti-Flag monoclonal antibodies and fluorescein isothiocyanate-anti mouse IgG. b, subcellular fractionation of 293T cells expressing wild type PKK. Cytosol was obtained from the post-nuclear supernatant of disrupted cells by ultracentrifugation. Membranes were extracted (TSM, Triton-soluble membranes) with 1% Triton X-100 in PBS. The remaining pellet was solubilized using SDS sample buffer (TIM, Triton-insoluble membranes). 4% of the total cytosol was loaded in the first lane, whereas 25% of the TSM and TIM were loaded in the adjacent lanes. An anti-Flag Western blot assay was performed. c, left panel, 293T cells transfected with wild type (WT) PKK were lysed with 1% Triton X-100; 25% of the pellet and 2.5% of the supernatant (Lysate) were separated by SDS-polyacrylamide gel electrophoresis, and Flag-tagged PKK was visualized on a Western blot. **Right panel,** subcellular fractionation of 293T cells transfected with wild type or K51R PKK. Cells were separated into a cytosolic fraction and a pellet, which included all membranes as well as the nuclear pellet. 2% of the cytosol and 50% of the pellet were separated by SDS-polyacrylamide gel electrophoresis, and Flag-tagged PKK was visualized on a Western blot. d, a 1% Triton X-100 lysate of 293T cells transiently transfected with wild type PKK was immunoprecipitated, and half of the immunoprecipitate (IP) was subjected to lambda phosphatase treatment. A portion of the pellet was also treated with lambda phosphatase (left panel). Another experiment

**Fig. 7.** Post-synthetic generation of a phosphorylated form of PKK. a, pulse-chase analysis of transiently transfected 293T cells reveals that PKK is synthesized initially as a 100-kDa protein, which is subsequently converted into a slower migrating 110-kDa form (left panel). In a separate experiment, cells were labeled metabolically with \[^{35}\text{S}\]methionine for 10 min. After a 1-h chase, PKK was immunoprecipitated, and one-half of the precipitate was treated with lambda phosphatase (right panel). b, pulse-chase analysis of cells transiently transfected with wild type PKK or with the K51R mutant. See the legend to panel a and the text for details. A prominent background band that migrates faster than PKK is seen in panel a and also in panel b. It is seen in anti-Flag immunoprecipitates of metabolically labeled mock-transfected 293T cells.
A kinase dead K51R mutant form of PKK is also initially synthesized as a 100-kDa form but is not converted to the 110-kDa form seen with wild type PKK (Fig. 7b). Small amounts of slower migrating K51R PKK proteins do appear with time; these might result from poorly defined transphosphorylation events (Fig. 7b). PKK phosphorylation and conversion to the 110-kDa form depends at least in part on autophosphorylation. The delay in activating PKK suggests that the nascent enzyme might need to move to a specific location within the cell or that it may in some temporally influenced manner interact with an activating kinase or with some other regulator. We performed pulse-chase studies on PKK in cells co-transfected with PKCβI, and found that even with the co-expression of PKCβI, a similar delay was observed in the post-synthetic generation of the 110-kDa form of PKK (data not shown). Although PKK probably requires activation by a distinct regulator, which is most likely an activation loop kinase, overexpression of PKCβI does not influence the in vivo generation of the 110-kDa form of PKK.

**DISCUSSION**

We have identified and molecularly cloned a novel serine/threonine kinase, PKK, which physically associates with the catalytic domain of PKCβI. This interaction was observed in the context of a two-hybrid assay using recombinant fusion proteins in vitro and in transfected cells in vivo. The interaction in the two-hybrid system suggests that PKCβI and PKK interact directly, and it is possible that PKK may represent a substate of PKCβI and also of other members of the PKC family. The C-terminal portion of PKK is phosphorylated by PKCβI in vitro. We have no evidence to suggest that PKK is actually phosphorylated by PKCβI in vivo. We have also been unable to demonstrate that PKCβI influences the catalytic activity of PKK.

The cloning of a human ankyrin repeat-containing kinase based on its association with PKC δ has been also recently described (27). This kinase, DIK (for PKC δ-interacting kinase), is identical to human PKK. DIK was shown to interact with the catalytic domain of PKCδ, but PKCδ does not phosphorylate DIK. It is unclear whether there is any physiological relevance to the interaction of PKK with PKCβI or PKCδ. Although there is some overlap in the expression patterns of PKCβ and PKK during embryogenesis, there are clearly developing tissues in the context of a two-hybrid assay using recombinant fusion proteins in vitro and in transfected cells in vivo. The interaction in the two-hybrid system suggests that PKCβI may influence the phosphorylation of the activation loop of PKK. These results suggest that PKK may need to interact with an activation loop kinase or some other regulatory molecule in the cell, or to alter its subcellular localization, in order to be activated. Because the N-terminal catalytic domain of PKK is not phosphorylated by PKCβI, it is likely that PKCβI does not directly influence the phosphorylation of the activation loop of PKK. The activation loop of PKK contains a Ser-X-X-Ser motif, which suggests that PKK is related to MAP kinase kinases and that it may be also activated in vivo by a MAP kinase kinase kinase. Studies are currently in progress to identify the MAP kinase kinase kinase that may be required for the activation of PKK.

The significance, if any, of the 112-kDa hyperphosphorylated, Triton X-100-resistant form of PKK is unclear. This fraction may potentially represent proteins that are in lipid rafts or proteins that are tightly bound to cytoskeletal components. The availability of antibodies that can be used to immunoprecipitate endogenous PKK would facilitate the examination of the biological significance of this form of PKK.

The expression of PKK is very tightly regulated during development. This speaks to an important developmental role for this kinase. In preliminary studies in which we targeted PKK K51R to developing lymphocytes, early B and T cell development is significantly compromised. The defect in B cell development observed in these preliminary studies is quite distinct from the peripheral B cell defect seen in mice lacking PKCβI (5). Studies are in progress to determine whether this kinase plays a role in early embryonic development.

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Protein Kinase C-associated Kinase (PKK), a Novel Membrane-associated, Ankyrin Repeat-containing Protein Kinase

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