Cloning and Characterization of a Novel RING Finger Protein That Interacts with Class V Myosins*

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We have identified a novel protein (BERP) that is a specific partner for the tail domain of myosin V. Class V myosins are a family of molecular motors thought to interact via their unique C-terminal tails with specific proteins for the targeted transport of organelles. BERP is highly expressed in brain and contains an N-terminal RING finger, followed by a B-box zinc finger, a coiled-coil (RBCC domain), and a unique C-terminal β-propeller domain. A yeast two-hybrid screening indicated that the C-terminal β-propeller domain mediates binding to the tail of the class V myosin myr6 (myosin Vb). This interaction was confirmed by immunoprecipitation, which also demonstrated that BERP could associate with myosin Va, the product of the dilute gene. Like myosin Va, BERP is expressed in a punctate pattern in the cytoplasm as well as in the neurites and growth cones of PC12 cells. We also found that the RBCC domain of BERP is involved in protein dimerization. Stable expression of a mutant form of BERP lacking the myosin-binding domain but containing the dimerization domain resulted in defective PC12 cell spreading and prevented neurite outgrowth in response to nerve growth factor. Our studies present a novel interaction for the β-propeller domain and provide evidence for a role for BERP in myosin V-mediated cargo transport.

The RING finger is a novel type of zinc-binding motif found in proteins involved in signal transduction pathways, gene regulation, and cellular differentiation (1). Members of the “RING-B-box-coiled-coil” (RBCC) subgroup of RING finger proteins have an N-terminal RING finger followed by one or two additional zinc-binding domains (B-box motifs), a leucine coiled-coil region, and a variable C-terminal domain (1, 2). Some RBCC proteins, including PML (3), TIF1 (4), KAP-1 (5), Rpt-1 (11), Staf50 (12), and HT2A (13), have only a single B-box, while MID1 (Fxy) gene (6, 7), possess an additional zinc-binding domain (B-box). Other RBCC proteins, including XNF7 (8), RFP (9), SS-A/Ro (10), Rpt-1 (11), Staf50 (12), and HT2A (13), have only a single B-box. RBCC proteins play key roles in regulating gene expression and cell proliferation. Opitz G/BBB syndrome, a defect of midline development, is due to mutations in an RBCC gene spanning the pseudoautosomal boundary (6, 7). XNF7 is a maternal expressed cytoplasmic protein that moves to the nucleus during mid-blastula transition, where it may regulate mitosis and dorsal ventral patterning of the embryo (8). Chromosomal translocations that result in the fusion of the RBCC domain to other gene products are frequently oncogenic (1, 3). For example, fusion of the PML RBCC domain with retinoic acid receptor-α underlies acute promyelocytic leukemia (3).

Another family of RING finger proteins is directly involved in the regulation of vesicular transport. The yeast RING finger protein Vac1 has been implicated in the regulation of vesicle docking and fusion via interactions with members of the synapton and Sec1 family (14). EEA1 and Hrs are early endosome-associated RING finger proteins (15, 16). Neurodap1 has been implicated in protein sorting from the Golgi to the postsynaptic density in neurons (17). Rlmi1 is a neuron-enriched RING finger protein localized to presynaptic active zones and has been implicated in neurotransmitter exocytosis (18). Finally, ARD1 is an RBCC protein that acts as a GTPase similar to the ARF family of proteins involved in the regulation of vesicular trafficking (19).

In this work, we report the identification of BERP, a novel member of the RBCC group of RING finger proteins that directly associates with the tail region of myosin V. The unconventional class V myosins are thought to interact via their C-terminal tails with specific proteins for the targeted transport of organelles within cells. BERP contains several protein-protein interaction motifs, making it a suitable adaptor protein for mediating myosin V cargo transport. Both BERP and myosin V are expressed with a punctate pattern in the cytoplasm and neurites of PC12 cells, a pattern characteristic for many organelle-associated proteins. Our results show that PC12 cells expressing a truncated form of BERP (lacking the C-terminal myosin-binding domain) do not respond to nerve growth factor with neurite outgrowth. These observations suggest that BERP may cooperate with myosin V in process outgrowth and the regulation of organelle transport.

EXPERIMENTAL PROCEDURES

Molecular Biology—A BERP cDNA was generated from rat cerebellar mRNA during a search for nucleotide-binding proteins, using the degenerate PCR1 forward primer 5′-TTI GCC TGG (T/C) TGG (T/C) TGG AC-3′ and reverse primer 5′-AAI ACC CA(G/A) CA(T/A) A(C/ A) A(G/A/G/A) AA-3′. The resulting PCR product was subcloned into pCRII (Invitrogen), providing a 32P-labeled probe for screening a rat brain cDNA library. A full-length cDNA was obtained and sequenced. The first ATG codon (position 221) had a Kozak consensus sequence, and a potential poly(A) signal was present in the 3′-noncoding region. For Northern analysis, total RNA was extracted using the single-step method, and a 1200-base pair EcoRI fragment of the 3′-end of the rat

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*This work was supported by the Medical Research Council of Canada. The costs of publication of this article were defrayed in part by the payment of page charges. This article must therefore be hereby marked "advertisement" in accordance with 18 U.S.C. Section 1734 solely to indicate this fact. The nucleotide sequence(s) reported in this paper has been submitted to the GenBankTM/EBI Data Bank with accession number(s) AF036255.

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1 The abbreviations used are: PCR, polymerase chain reaction; ABP, actin-binding protein; NGF, nerve growth factor.
BERP cDNA was used to generate 32P-labeled probes by random priming (Life Technologies, Inc.).

Yeast Two-hybrid Analysis—To identify proteins interacting with BERP, the yeast two-hybrid system was used to screen a rat brain cDNA library subcloned into pGAD10 containing the GAL4 activation domain (CLONTECH MATCHMAKER system). Different regions of BERP were subcloned into pAS2-1 in frame with the GAL4 DNA-binding domain as baits, and target sequences were subcloned into pACT2 in frame with the GAL4 activation domain (see Table I). Plasmids from positive clones were identified by sequencing.

Cell Transfection—To insert the FLAG sequence at the N-terminal end of BERP, the BERP cDNA was digested with BstEII/PpuMI, and a forward primer containing the BstEII site followed by a FLAG sequence before the first ATG codon (5'-AACTGGTCACCGCCACCATGGACTACAAGGACGACGATGACAAGATGGCAAAGAGGGAGGAC-3') and a reverse primer including the PpuMI site (5'-AGCCCAGTCTCAGTGCTCCT 3') were used to generate a PCR product, which was subcloned into the BstEII/PpuMI site of the digested BERP cDNA. The BERP cDNA containing the FLAG sequence was then subcloned into XhoI/BamHI sites of pcDNA3. This FLAG-tagged, full-length BERP cDNA was digested with EcoRI and religated to give a construct coding for an N-terminal FLAG followed by the first 485 amino acids of BERP (NBERP). A histidine-tagged myr6 C-terminal region was amplified by PCR using the myr6 cDNA obtained from the yeast two-hybrid screening with the forward primer 5'-ACCATGCATCACCATCACCATCACGACCAAGC-CATGCAGGAT-3' and the reverse primer 5'-GAAAACAAGCATCCTCA-3'.

PC12 cells were cultured on poly-L-lysine-coated plates in Dulbecco's minimal essential medium supplemented with 10% horse serum and 5% fetal calf serum and plated at ~30–50% confluency 1 day prior to

Fig. 1. Predicted amino acid sequence and domains of BERP. a, deduced amino acid sequence of BERP. Conserved cysteines and histidines of the RING finger and B-box motifs are in blue. The B-box is followed by a coiled-coil motif (dashed underline) and an ABP-like domain (solid underline). The C-terminal region of BERP (indicated with an arrow) consists of seven repeats similar to WD or kelch repeats. b, diagram of the various domains present in BERP. c and d, alignments of the RING finger and B-box domains, respectively, with those present in other proteins demonstrate the conserved zinc-binding ligands (blue). e, the RBCC domain is followed by an ~130-amino acid domain (ABP-like repeat) with a predicted β-sheet structure that is present in 24 repeats in ABP-280 and in six repeats in ABP-120. Conserved amino acids in the ABP-like repeat are in yellow.

f, the C-terminal region of BERP consists of six repeats of a novel domain very similar to the WD repeats present in G protein β-subunits (Gβ). These repeats are predicted to form a β-propeller fold with six blades. Each repeat is ~45 amino acids long and contains four β-strands (a–d, underlined) separated by loops. Note that the d-strand of the last repeat is provided by the N terminus of the domain. Amino acids present in at least three of the repeats are shown in blue.

g, the RBCC domain is followed by an 130-amino acid domain (ABP-like repeat) with a predicted β-sheet structure that is present in 24 repeats in ABP-280 and in six repeats in ABP-120. Conserved amino acids in the ABP-like repeat are in yellow.
transfection with 10–15 μg of DNA using the calcium phosphate method. To develop stable PC12 cell lines, fresh medium containing 500 μg/ml G418 (Life Technologies, Inc.) was added after 3 days for selection. This medium was changed every 3–4 days, and surviving clones were collected and individually plated after 2 weeks in medium containing 200 μg/ml G418. Western blot analysis using anti-FLAG antibodies confirmed the expression of proteins of the predicted size.

**Immunocytochemistry**—Cultured cells were fixed for 1 h with 4% paraformaldehyde, rinsed with phosphate-buffered saline, incubated overnight at 4 °C with primary antibodies in phosphate-buffered saline containing 0.3% Triton X-100 and 2% normal goat serum, and then processed for ABC immunoperoxidase staining (ABC Elite, Vector Laboratories, Burlingame, CA) using diaminobenzidine with nickel ammonium sulfate.

**Western Blotting and Immunoprecipitations**—Rabbits were immunized with a peptide corresponding to the last 9 amino acids of BERP conjugated to keyhole limpet hemocyanin. Anti-BERP antibodies were affinity-purified using the C-terminal BERP peptide linked to activated thiol-Sepharose beads (Amersham Pharmacia Biotech). For immunoprecipitation, brain tissues were homogenized in lysis buffer (50 mM Tris, 250 mM NaCl, 1 mM EDTA, 10 μg/ml aprotinin, 1 μg/ml leupeptin, 1 μg/ml pepstatin, and 10 mM phenylmethylsulfonyl fluoride) containing 0.1% Nonidet P-40, whereas cell cultures were lysed in 500 μl of lysis buffer containing 1% Triton X-100 for 20 min at 4 °C with agitation. Lysates were incubated with antibodies for 1–2 h on ice, precipitated with protein A-Sepharose beads, and boiled in 40 μl of SDS sample buffer to extract immunoprecipitated proteins. Western blots were detected with ECL (Amersham Pharmacia Biotech).

**RESULTS**

**BERP Is a Novel RING Finger Protein with a C-terminal β-Propeller**—A cDNA encoding a novel, brain-expressed RING finger protein (BERP) was discovered in a reverse transcription-PCR analysis of rat brain mRNA. The full-length BERP cDNA was then obtained by screening a rat brain cDNA library using this PCR product as a probe (Fig. 1a). Data base BLAST searches showed that this cDNA encoded a novel protein containing three motifs present in the RBCC subgroup of RING finger proteins (2). Fig. 1b indicates the various motifs present in BERP. The N-terminal amino acids 22–62 compose the RING finger, and sequence alignments of various RING fingers show the conserved zinc-binding ligands (Fig. 1c). Following the RING finger is the B-box, another form of zinc finger (Fig. 1d), which in turn is followed by a coiled-coil domain (20).

After the RBCC motif, BERP contains a domain of ~130 amino acids (amino acids 290–420) that is 41% identical to a repeated domain present in the actin-binding proteins ABP-120 and ABP-280 (Fig. 1e) (21, 22). These repeats are thought to mediate protein-protein interactions (21, 22). The C terminus of BERP is characterized by the presence of six repeats of a novel domain very similar to kelch or WD repeats (Fig. 1f) (23–25). A secondary structure analysis using prediction-based threading (26) is consistent with this domain forming a circular β-propeller with six blades, similar to that described for other WD and kelch proteins (27, 28).

Northern analysis indicated that the BERP transcript is ~3 kilobases in size and is highly expressed in the brain. Moderate levels of BERP expression were seen in the lung, whereas the liver, kidney, and heart expressed only very low levels of BERP mRNA (Fig. 2). In the brain, expression was highest in the cerebellum, although transcripts were detected in all brain regions examined (Fig. 2).

**BERP Oligomerization**—The RBCC domain may serve as a protein oligomerization motif (1). To determine if the BERP RBCC domain is involved in BERP oligomerization, we first employed the yeast two-hybrid system using various BERP constructs subeloned into the pAS2-1 vector, which contains the GAL4 DNA-binding domain, and the pACT2 vector, which contains the GAL4 activation domain, to generate baits and targets, respectively. As shown in Table I, BERP sequences that contain the N-terminal RBCC domain (amino acids 1–372) were able to interact. Deletion of amino acids 263–372 did not abolish this interaction, indicating that the full coiled-coil domain is not necessary and that the first 262 amino acids, which include coils 1 and 2, are sufficient for BERP oligomerization.

A biochemical method was used to confirm this interaction. A truncated form of BERP (NBERP, amino acids 1–485) that contains the RBCC domain but lacks the C-terminal β-propeller domain co-immunoprecipitated with full-length BERP from lysates of HEK-293 cells transfected with BERP and NBERP (Fig. 3). Thus, both the yeast two-hybrid analysis and immunoprecipitation experiments showed that BERP constructs containing the RBCC domain can associate together, supporting a role for the RBCC motif in protein homodimerization in vivo.

**BERP, Unlike HT2A, Does Not Interact with the Human Immunodeficiency Virus Regulatory Protein Tat**—HT2A is a human RBCC protein that shows significant overall homology to BERP and contains a similar C-terminal β-propeller domain. It was identified in a yeast two-hybrid screening for proteins that interact with the human immunodeficiency virus protein Tat, and the last 120 amino acids of HT2A were sufficient for Tat binding (13). Because of the high similarity between the C-terminal β-propeller domains of BERP and HT2A (~30% identity), we examined whether BERP could also interact with the human immunodeficiency virus Tat protein. Several baits that contained BERP C-terminal sequences were used (Table 1). The C-terminal region of HT2A was used as a positive control and interacted strongly with Tat, as reported by Fridell et al. (13). In contrast, none of the BERP baits showed a positive interaction with Tat. This suggests that HT2A-specific sequences that are absent from BERP are involved in the interaction between HT2A and Tat. The cellular proteins that interact with HT2A have not yet been identified.

**BERP Interacts with the Tail Domain of Class V Myosins**—The yeast two-hybrid system was used to identify proteins that may interact with BERP. From 15 × 10^6 transformants, 10 positive clones were isolated when a bait that included amino acids 383–744 of BERP was used for screening. Two of these showed specific interaction with the C-terminal domain of BERP (Table 1). Sequencing analysis indicated that both positives represented the same C-terminal region (the last 641 amino acids) of a recently identified unconventional class V myosin, myr6 (myosin Vb) (29). Various BERP C-terminal baits were able to interact with the myosin Vb C-terminal region (Table 1). A bait comprising only the last 124 amino acids of BERP was sufficient.
The interaction between BERP and myosin Vb was confirmed by immunoprecipitation. Anti-FLAG antibodies could immunoprecipitate FLAG-tagged, full-length BERP together with a His-tagged myosin Vb C-terminal tail domain from lysates of HEK-293 cells cotransfected with these constructs (Fig. 4). Thus, in both yeast and mammalian cells, BERP can directly interact with the tail domain of the mammalian class V myosin myr6.

The protein encoded by the dilute locus, myosin Va, is a class V myosin that is highly expressed in the brain (30) and is closely related to myr6 (78% identity) (29). To determine if BERP could also associate with myosin Va, antibodies against this protein (31) were used for immunoprecipitation experiments using rat brain extracts (Fig. 5). Affinity-purified anti-BERP antibodies that recognize endogenous BERP from brain extracts were used for detection of immunoprecipitated BERP (Fig. 5A). Endogenous BERP co-immunoprecipitated with endogenous myosin Va from cortex and cerebellar extracts (Fig. 5B). These results indicate that BERP is an in vivo partner of class V myosin in brain tissue.

**Table I**

Yeast two-hybrid analysis of BERP interactions

| Bait          | Target       | RBCC1 | RBCC2 | ABPL  | CBERP2 | HT2A  | myr6 | pTD1 |
|---------------|--------------|-------|-------|-------|--------|-------|------|------|
| RBCC1 (aa 1–372) | His⁺ and and blue |       |       |       |        |       |      |      |
| RBCC2 (aa 1–282) | His⁺ and and blue |       |       |       |        |       |      |      |
| ABPL (aa 285–495) | blue        |       |       |       |        |       |      |      |
| CBERP1 (aa 383–744) | blue        |       |       |       |        |       |      |      |
| CBERP2 (aa 459–744) | blue        |       |       |       |        |       |      |      |
| CBERP3 (aa 620–744) | blue        |       |       |       |        |       |      |      |
| TAT           | blue        |       |       |       |        |       |      |      |

aa, amino acids.
cells, indicating that the cytoplasmic localization of BERP is not unique to PC12 cells (data not shown). After NGF treatment, both myosin Va (Fig. 6C) and full-length BERP (Fig. 6D) were present in puncta in the cell cytoplasm as well as in the processes and growth cones that had extended from these cells.

Myosin Va appears to play a role in neurite extension and growth cone development (32). Since BERP interacts with the tail of the class V myosins, we examined the effect of expression of NBERP, a truncated form of BERP that lacks the myosin-binding domain, on the growth and differentiation of PC12 cells. For this purpose, stable cell lines expressing FLAG-tagged NBERP were generated. Western blot analysis using anti-FLAG antibodies confirmed the expression of proteins of the predicted sizes (data not shown). Significantly, cells expressing NBERP were small and rapidly dividing and formed tightly clustered aggregates. This phenotype and staining pattern were observed in 10 independently derived cell lines expressing NBERP.

We next examined how the expression of BERP and NBERP affected the differentiation of PC12 cells induced by NGF. The transfected PC12 cell lines were treated with NGF for 7 days and then fixed and immunostained. Neurite outgrowth was monitored either with phase-contrast microscopy (Fig. 7, a–c) or after immunostaining for myosin Va, which stains cell bodies and neurites (Fig. 7, d–f). Cell lines transfected with empty vector (Fig. 7, a and d) or full-length BERP (Fig. 7, b and e) as well as untransfected PC12 cells (data not shown) responded to NGF treatments, ceased dividing, and developed long processes. Surprisingly, none of the cell lines expressing NBERP responded to NGF with differentiation and the production of neurites (Fig. 7, c and f). Instead, these cells continued to proliferate rapidly like the NBERP cell lines that had not received NGF treatment.

**DISCUSSION**

We have identified a novel RING finger protein (BERP) that interacts directly with class V myosins. BERP binding to the C-terminal tail of the mammalian class V myosin (myr6) was demonstrated in the yeast two-hybrid system and confirmed by immunoprecipitation. BERP was also found to associate with the closely related myosin Va in brain extracts. Expression of a truncated form of BERP lacking this myosin-binding domain resulted in abnormal cell aggregation and defective cell spreading. Furthermore, expression of this truncated form prevented NGF-induced neurite outgrowth in PC12 cells.

BERP belongs to the RBCC subgroup of RING finger proteins, which contain a second zinc finger known as the B-box followed by a coiled-coil domain (1, 2). The RBCC domain may mediate protein oligomerization, and RBCC proteins are often found in large complexes, perhaps acting as scaffolding elements (33). Using the yeast two-hybrid system, we have found that the RBCC domain is also necessary for BERP dimerization. Cell transfection and immunoprecipitation demonstrated that full-length BERP associates with a truncated form containing the RBCC domain but lacking C-terminal sequences. These results provide direct evidence for a role for the RBCC domain in protein homo-oligomerization. Because the RBCC structure is highly conserved in a number of proteins, this domain may also allow various members of the RBCC group to physically interact.

Immediately after the RBCC motif, BERP contains a domain highly similar to a repeating domain found in the actin-binding proteins ABP-120 and ABP-280 (21, 22). These repeats each form an immunoglobulin-like fold and mediate protein–protein interactions with the high affinity IgG receptor, β2-integrin, the stress-activated protein kinase activator SEK-1, and the presenilins (34). The high similarity between the repeats pres-
BERP, a RING Finger Protein Partner of Myosin V

The C terminus of BERP is characterized by the presence of six repeats of a novel domain very similar to kelch or WD repeats (23, 24). Secondary structure predictions (26) are consistent with this domain forming a circular β-propeller with six blades (27, 28). In BERP, the variable N-terminal portions of each repeat, together with the d-strands (Fig. 1f), would be displayed on the outer surface of each blade and provide sites for protein-protein interactions (27, 28). This structure is found in a variety of proteins involved in signal transduction, RNA processing, gene regulation, vesicle fusion, and cytoskeletal assembly (24). Our work identifies a novel function for the β-propeller domain. We show that the β-propeller domain of BERP directly interacts with the tail region of the molecular motor myosin V. Recently, a β-propeller domain has also been identified in clathrin, a vesicle coat protein regulating vesicle transport that interacts with multiple partners, including the AP1 and AP2 sorting adaptor complexes (25). Similarly, the various domains found in BERP, including the β-propeller, the RBCC domain, and the ABP-like repeat, may mediate interactions with various proteins, allowing cargo transport via a myosin V-mediated pathway.

Data base searches identified sequences very similar to the C-terminal β-propeller of BERP in a recently identified mycobacterial serine-threonine protein kinase, Mbk (35), and in two other mammalian RBCC proteins, HT2A (13) and KIAA0517 (36). Indeed, BERP shows an overall similarity of ~35% to HT2A and is 65% identical to KIAA0517, a protein identified through random sequencing of large protein-coding sequences from brain (36). The cellular functions of these proteins are not yet known; however, the Arabidopsis protein COP1, which acts as a photomorphogenic repressor, also has an N-terminal RING finger, a coiled-coil domain, and a C-terminal β-propeller, although it lacks a B-box (37). Together, these molecules form a novel family of RING finger-β-propeller proteins.

The class V myosins are among the most well characterized of the unconventional myosins, and there is evidence that they participate in organelle trafficking (38). Mutations in yeast myosin V (Myo2p) result in vesicle accumulation (39). In melanocytes, myosin Va associates with melanosomes, allowing their delivery along the dendrites to the keratinocytes (40). Myosin V has also been suggested to function as an actin-based organelle motor in neurons. Indeed, myosin Va binds to synaptic vesicles through interactions with synaptobrevin and synaptophysin (41). Recently, it was shown that myosin Va is concentrated in a punctate pattern in organelle-rich regions of the growth cone (31). These data are consistent with an important role for class V myosins in the transport of specific organelles to sites of outgrowth. Indeed, our results show that both BERP and myosin Va are expressed with a punctate pattern in the neurites and growth cones of PC12 cells, a localization characteristic of organelle-associated proteins (15, 17, 31, 39, 40).

A role for myosin V in regulating cell spreading and motility has also been suggested (42), and inactivation of myosin Va using the chromophore-associated laser technique resulted in defects in filopodial extension of neuronal growth cones (32). The phenotype observed in PC12 cells expressing NBERP indicates a defect in cell spreading that may have resulted from disruption of the interaction between endogenous BERP and myosin V. Furthermore, cells expressing NBERP continued to rapidly proliferate and failed to generate neurites in response to NGF. If BERP interacts directly with myosin V, one would predict that a dominant-negative interference with BERP function would result in a phenotype reflecting a disruption in myosin V function. Indeed, expression of an N-terminal fragment of COP1 containing the RING finger and coiled-coil, but...
lacking the C-terminal β-propeller, caused a dominant-negative phenotype in transgenic plants (37). Our data demonstrated that the BERF RBCC domain mediates protein dimerization and that PC12 cells do express endogenous BERF. Thus, it is likely that NBERF can act in a dominant-negative manner, dimerizing with wild-type BERF and suppressing its function. Together, these data indicate that BERP interactions with class V myosins may be important in organelle transport and neurite outgrowth.

Acknowledgments—We thank Terry Snutch for providing the rat brain cDNA library, Brian Cullen for the HT2A and TAT constructs, and Paul Bridgman for the antibody to rat myosin Va. Tim Murphy provided assistance with confocal microscopy, and Chris Bladen, Rouzbeh Shooshtarian, and Dorota Kwasnicka provided other technical assistance.

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J. Biol. Chem. 1999, 274:19771-19777.  
doi: 10.1074/jbc.274.28.19771

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