Molecular Structure of Microtubule-associated Protein 2b and 2c from Rat Brain*

(Received for publication, July 24, 1990)

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The vertebrate nervous system is composed of neurons with a large variety of different morphologies. One class of cytoskeletal elements which appear to play a crucial role in determining the shape of nerve cells are the microtubules and their associated proteins, called MAPs (1). The latter appear to be important for both the assembly and stability of microtubules (2), as well as for the overall shape and plasticity of neuronal processes (3-5). This appears to be accomplished by varying the affinities of MAPs for microtubules during neurite outgrowth (2), as well as for the overall shape and plasticity of neuronal processes (3-5). This appears to be accomplished by varying the affinities of MAPs for microtubules during neurite outgrowth (2), as well as for the overall shape and plasticity of neuronal processes (3-5).

Full length cDNA clones encoding microtubule-associated proteins (MAP) 2b and 2c from rat brain have been isolated and sequenced. The cDNA fragments spanning the coding regions for both MAP2b and MAP2c were assembled and expressed in Escherichia coli. The mobility of these bacterial expressed proteins in sodium dodecyl sulfate gels is identical to that of MAP2b and MAP2c from rat brain. The protein sequence of rat MAP2b has been compared to the full length sequence from mouse and the partial sequence from human high molecular weight MAP2. This comparison has revealed that MAP2b is composed of several highly conserved domains flanked by domains with extensive sequence divergence. Two of the conserved domains, found either at the NH2 or COOH terminus, overlap with the binding domain for the regulatory subunit of the cAMP-dependent protein kinase II and the microtubule-binding domain, respectively. A third homologous domain of unknown function lies in a central region of MAP2b. Secondary structure prediction suggests that the portion of MAP2b which extends from the microtubule surface is composed of an extensive number of α-helices separated by small turns which may account for the extended yet flexible structure of MAP2b. Interestingly, the 4000-base pair deletion from the middle of MAP2b which generates MAP2c not only removes these helices, but also this third highly conserved MAP2b domain.

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In this report we describe the full length cDNA cloning, sequencing, and expression in Escherichia coli of both rat brain MAP2b and MAP2c. We also present an analysis of their primary and secondary structures, and a model describing the role these two proteins might play in determining dendritic plasticity. In addition, we have compared the amino acid sequence of the entire rat MAP2b to the full length mouse high molecular weight MAP2 (12) as well as a partial human high molecular weight MAP2 sequence (16, 17). This comparison has revealed that MAP2b is composed of several highly conserved domains, some of which overlap with previously identified functional domains, and others suggesting the existence of new functional domains.

EXPERIMENTAL PROCEDURES

Materials—Restriction enzymes were purchased from Pharmacia LKB Biotecnology Inc. and calf intestinal phosphatase from Boehringer Mannheim.

Isolation of cDNA and Genomic Clones—Two cDNA libraries were assembled, one from a postnatal day 5 rat brain poly(A+) RNA, described previously (9), and the second from glioma C6 cell mRNA. This second library was constructed in an identical manner as the rat library (9) in λgt11. The rat liver genomic library, constructed in EMBL3 (Stratagene) was a generous gift from G. Schuetz (Deutscher Krebs Forschung Zentrum, Heidelberg). Genomic and cDNA clones were identified by plaque hybridization as described by Maniatis (45). Positive clones were plaque purified and cDNA inserts subcloned into pUC18 (46) before sequencing with T7 polymerase (Pharmacia) as described by the manufacturers.

Expression of MAP2b and MAP2c in E. coli—cDNA clone 1H11 (Fig. 1) was subcloned into M13 and the nucleotides around the ATG

* This work was supported by the Federal Government of Germany (Bundesministerium für Forschung und Technologie). 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 GenBank™/EMBL Data Bank with accession number(s) X58482.

† This work forms part of a doctoral thesis.

‡ The abbreviations used are: MAPs, microtubule-associated proteins; SDS, sodium dodecyl sulfate.
of MAP2 were changed to create a NdeI site by site-directed mutagenesis (45). MAP2c was assembled by taking the NdeI-PstI fragment from 14bII and the &I-&oRI fragment from 38a (Fig. 1) and ligating them downstream of the T7 RNA polymerase promoter into the N&I-EcoRI cut pRKl72 vector (48). MAP2b was constructed by first assembling the NdeI-EcoRI fragment from 14bII and an &oRI-Not1 fragment from 14bI and 19a. Constructs were transfected into E. coli strain BL21DE3 (49) and grown in Luria broth + ampicillin (10 &ml) to an A 1,000 of 0.6. IsopropyI-l-thio-&D-galacto-pyranoside was added to 1 mM and the cells grown for a further 30 min. Cells were harvested and resuspended in 1 × Laemmli SDS sample buffer before boiling for 5 min. Samples were run on 3-15% SDS-polyacrylamide gels and the proteins blotted onto nitrocellulose (9).

Rat brain S1 supernatants and phosphatase treatments were performed as described by Garner et al. (20).

MAP2c-specific Antibodies—Rabbit polyclonal antibodies against the 8-amino acid peptide spanning the MAP2c junction (Fig. 3) were produced by synthesizing a 10-amino acid peptide containing cysteines on both ends (Milligan, Burlington MA), cyclizing it, and coupling the peptide to thyroglobulin (47). This conjugate was emulsified with Freund's complete adjuvant and injected subcutaneously into New Zealand white rabbits. Booster injections were performed at 2-week intervals using Freund's incomplete adjuvant. Antibodies specific for the MAP2c were affinity purified with E. coli expressed MAP2c immobilized on nitrocellulose by Western blotting. The antibody was bound in phosphate-buffered saline, 2% fetal calf serum for 2 h at room temperature. After washing 3 × 10 min with phosphate-buffered saline, the antibody was eluted in 0.2 M trichloroacetic acid, pH 7.0, for 1 h and then dialyzed against phosphate buffered saline before use.

Computer Analysis—Nucleotide sequencing, amino acid sequence homologies, and secondary structure predictions using the algorithm of Garnier et al. (22) and Chou-Fassman (21) were analyzed with the DNA-Star computer program (DNA-Star Inc. Madison, WI.).

RESULTS
cDNA Clones Encoding Rat Brain MAP2b—cDNA clones encoding pieces of MAP2b and MAP2c were initially isolated from a random primed λ gt11 expression library screened with MAP2 antibodies (9). The cDNA inserts from two of these clones, 38a and 19a (Fig. 1), were then used to rescree the same library to isolate a series of overlapping clones. The layout of these clones and the strategy used to determine 100% of their sequences from both strands is shown in Fig. 1.

One set of these clones (14b, 19a, and 4.2) contains an open reading frame encoding a protein of 1830 amino acids (Fig. 4). The methionine at position 1 conforms to the eukaryotic translation start site sequence (18) and is preceded by two in-frame stop codons (19). The molecular weight of the encoded protein is calculated to be 200,000. This is about 80 kDa smaller than that predicted for high molecular weight MAP2 on SDS gels.

In order to resolve this discrepancy, we assembled this group of clones and expressed the encoded protein in E. coli. As shown in Fig. 2, lane 1, the E. coli expressed protein has an apparent molecular weight on SDS gels of 280,000. A comparison of the mobility of the E. coli expressed protein to that of MAP2b and MAP2b from adult rat brain on SDS gels (Fig. 2, lane 2) demonstrates clearly that our E. coli expressed protein has an identical mobility to MAP2b (Fig. 2, lane 3). It would therefore appear that this collection of clones encode MAP2b and not MAP2a.

Genomic and cDNA Clones Encoding MAP2c—The cDNA sequence analysis of both strands of clone 38a revealed that this clone was composed of nucleotide sequences found at both the 5' and 3' end of the first set of clones (Fig. 1). The deduced amino acid sequence from 38a contained an open reading frame which was identical with amino acids 50-152 and the last 316 amino acids of the MAP2b sequence (underlined sequence in Fig. 4).

Since this and earlier data (9) suggested that clone 38a might encode a portion of MAP2c, we sought to answer two questions. First, does 38a actually encode MAP2c (see next
section), and second do MAP2c and MAP2b contain identical amino-terminal sequences? We were able to answer the second of these two questions by two separate approaches. The first was to isolate and sequence cDNA clones that extended upstream from the 5′ end of 38a. This was accomplished by screening a glioma C6 random primed cDNA λ gt11 expression library with clone 38a. This cell line was chosen since we had previously observed that C6 cells express only MAP2c and a 6-kilobase mRNA thought to encode MAP2c (9). A 500-base pair clone, C6.11, was identified (Fig. 1) which overlapped with the 5′ end of 38a and extended the sequence of 38a by 350 nucleotides in the 5′ direction. The sequence of this clone was found to be identical with the first 500 nucleotides of our MAP2b clones and the first 150 nucleotides from the 5′ end of 38a (Fig. 1). The sequence included the two in-frame stop codons followed by the initiating methionine found in MAP2b.

This suggested that both MAP2b and MAP2c have an identical amino terminus, however, to verify this, we also isolated genomic clones from a rat liver genomic library constructed in an EMBL3 λ vector which hybridized to a 5′ EcoRI fragment from MAP2b, called 14bII (Fig. 1), and the 5′ end of 38a. Several clones were found that hybridized to 14bII. When mapped, it was found that the first 500 base pairs of MAP2b were encoded on 5 exons spanning 50 kilobases of genomic DNA (data not shown). One of these exons was found to be 200 nucleotides long and to be colinear with both the proposed MAP2b initiating codon and the 5′ end of 38a.

**Fig. 3.** Structural relationship of MAP2b to MAP2c. MAP2b is 1830 amino acids in length while MAP2c consists of 467 amino acids. The solid regions in both MAP2b and 2c represent the 3 18-amino acid repeats that make up a part of their tubulin-binding domains. MAP2c is created by an alternative splicing of MAP2b that deletes 1362 amino acids from its middle. The peptide sequence spans the MAP2c splice site and was used to generate the rabbit anti-MAP2c antibody. The arrow in the middle of this peptide sequence represents the point of the MAP2c splice junction.

**Fig. 4.** Comparison of the rat brain MAP2b and 2c protein sequence to that of the mouse and human high molecular weight MAP2. The underlined sequence of MAP2b indicates the amino acids that compose MAP2c. The tubulin and regulatory subunit of the CaM-dependent protein kinase II binding domains are in boxes, as well as the proposed calmodulin-binding domain. Proposed CaM kinase and CaM-dependent kinase phosphorylation sites are indicated as overlines. Dashes indicate amino acids not present in a given sequence.
The identity of this protein was further corroborated by thyroglobulin, that spans the proposed splice junction identified postnatal day 7 rat brain Sl phosphatase-treated supernatant. Molecular Weight MAPZ-Presented in Fig. 4 is a comparison of the amino acid sequence from rat MAP2b to the full length sequence shows a 92% overall homology to the mouse and 76% calmodulin-binding site in MAP2b. This demonstrates that MAP2c is composed of identical sequences from the first 132 and last 316 amino acids of MAP2b.

Clone 38a Encodes MAP2c—Since the combined sequences from clone 38a and C6.11 predict a protein of about 43 kDa, whereas the predicted molecular mass of MAP2c was 70 kDa (20), we wished to verify that the protein encoded by these clones was MAP2c. This was accomplished by two separate approaches. In the first, we assembled the cDNA fragments predicted to encode MAP2c and expressed them in E. coli. In Fig. 2, we show Western blots stained with an anti-MAP2 monoclonal antibody comparing the mobility of our E. coli expressed protein to rat brain expressed MAP2c. As seen in lane 3, postnatal day 7 rat brain expresses several immunoreactive MAP2 bands running at 70 kDa. Since these multiple bands are due to protein phosphorylation, we pretreated our preparation with calf intestinal alkaline phosphatase overnight (lane 4). As observable in lane 5, our E. coli expressed construct has an identical mobility to the dephosphorylated MAP2c (lane 4) suggesting that our clones encode MAP2c.

The identity of this protein was further corroborated by immunizing rabbits with an 8-amino acid peptide, coupled to thyroglobulin, that spans the proposed splice junction identified in 38a (Fig. 3a). Sera from one of these rabbits reacted specifically with a 70-kDa protein on Western blots from postnatal day 7 rat brain S1 phosphatase-treated supernatant and from the E. coli expressed MAP2c (Fig. 2, lane 7) and not with MAP2b (lane 8). These data demonstrate that MAP2c is a 42.3-kDa protein which runs at 70 kDa on SDS gels and that it is missing 1363 amino acids present in the middle of MAP2b.

Comparison of Rat MAP2b to Mouse and Human High Molecular Weight MAP2—Presented in Fig. 4 is a comparison of the amino acid sequence from rat MAP2b to the full length amino acid sequence from mouse high molecular weight MAP2 described by Lewis et al., (12) and the partial human high molecular weight MAP2 (16, 17). The rat MAP2b sequence shows a 92% overall homology to the mouse and 76% to human. However, only tells part of the story since as seen in Fig. 4, the non-homologous amino acids are clustered and separated by domains of nearly complete homology. Interestingly, several of these highly conserved regions have recently been identified as domains involved in microtubule binding (amino acids 650–920) (11, 13) and in the binding of the regulatory subunit of CaM-dependent kinase (amino acids 75–125) (14, 15). Two additional highly conserved domains lie between amino acids 650 and 940 (see next section) and between amino acids 1370 and 1650 (Fig. 4). The latter domain has three very interesting features: first, it is very rich in proline; second it contains many of the proposed phosphorylation sites for both Ca2+/calmodulin (CaM)-dependent protein kinase type II (CaM kinase) and cAMP-dependent kinase; and third it contains a stretch of positively charged amino acids whose arrangement resembles the CaM-binding domains found in other proteins (Fig. 5). These characteristics, as well as the fact that it lies just 5’ of the microtubule-binding domain, suggest that this region is a hinge domain of MAP2b and 2c whose functional properties can be altered by phosphorylation.

Secondary Structure Prediction of MAP2b—Using two different computer programs which utilize the principles established by Garnier-Robson and Chow-Fassman (21, 22) to predict the secondary structure of proteins, we have analyzed the helix, coil, and turn distribution in MAP2b. These programs predict that MAP2b is composed of 12% turn, 62% helix, and 26% coil structure. However, as diagramed in Fig. 6 these predicted secondary structures are clustered into several domains. Beginning with the COOH terminus and ending with the MAP2c splice junction, one finds very little secondary structure predicted (Fig. 6). The 3’ end of this domain has previously been identified by several groups as the domain essential for microtubule binding (12, 23). Near the MAP2c junction is a region very rich in prolines which could be part of a hinge domain from which the remainder of MAP2b extends from the microtubule surface.

The next domain begins at the MAP2c junction and extends two-thirds of the distance to the NH2 terminus. This region is predicted to be composed of some 19 helices in a row separated by very short turns. In principle this domain might be predicted to be a very flexible rod. Adjacent to this is a sequence of amino acids, again with very little secondary structure followed by several more regions rich in helices before coming to the NH2 terminus. It should be noted that this second coil domain overlaps with the highly conserved domain spanning amino acids 650–920 as described earlier, as well as a second clustering of possible CaM and cAMP-dependent kinase sites (Figs. 4 and 6).

DISCUSSION

In this paper we have described the cDNA cloning, sequencing, structural characterization, and expression of two isoforms of MAP2. The cDNA clones spanning the rat brain high molecular weight MAP2 were found to encode a protein of 200,000. Although this is some 80 kDa smaller than previous estimates on SDS-polyacrylamide gels (24), it is in accord with estimates of 220 kDa from sedimentation equilibrium centrifugation and gel filtration experiments (25), as well as with the predicted molecular mass of the cloned mouse high molecular mass MAP2 (12). When clones encoding the high molecular weight rat brain MAP2b were assembled and expressed in E. coli, the encoded protein was found to be...
immunoreactive with previously characterized monoclonal antibodies against MAP2, as well as to co-migrate on SDS gels with rat brain MAP2b. This suggests that our clones encode MAP2b. However, it raises the question as to what structural modification of MAP2b generates MAP2a? Binder et al. (26) suggested that MAP2a may be generated from MAP2b by phosphorylation. However, we and others have never been able to generate MAP2b from MAP2a by phosphate treatment so that it is still not clear whether MAP2a is generated via some other post-translational modification or alternative splicing. A second set of clones were found to encode a 43-kDa protein which is composed of sequences from the 5' and 3' ends of the high molecular mass MAP2. Antibodies generated against an 8-amino acid peptide spanning the splice sequence (Fig. 2) in this protein were found to be immunoreactive with a 70-kDa MAP which co-migrates with MAP2c. The E. coli expressed form of this protein was also found to co-migrate on SDS-polyacrylamide gels with dephosphorylated rat brain MAP2c and was immunoreactive with known anti-MAP2 monoclonal antibodies demonstrating that these clones encode MAP2c.

A comparison of the protein sequences for MAP2b and MAP2c (Fig. 4) demonstrates that both proteins possess identical amino-terminal sequences which have recently been shown to bind the regulatory subunit of the CaM-dependent kinase (14, 15) and identical carboxy-terminal domains where both bind to microtubules. These data indicate that MAP2c is lacking the central domain of MAP2b thought to be involved in intra-cytoskeletal cross-bridging (44) and is generated as a result of alternative splicing of a primary MAP2 transcript (9).

Structural Analysis of MAP2b and MAP2c—Previous structural analysis of the high molecular mass form of MAP2 and the 65-kDa microtubule-associated protein by 1H NMR, 14C NMR, and circular dichroism (25, 27-29) suggested that these proteins contain very little secondary structure (7% a-helix and 90% unordered conformation) (25). Interestingly, when Woody et al. (27) studied the changes in flexibility of high molecular weight MAP2 and 7 before and after binding to microtubules by 1H NMR, they observed that whereas the binding of MAP2 to the microtubule surface had very little effect on its degree of flexibility, 7's flexibility decreased after binding. For MAP2c, the measured flexibility was shown to lie in its long extended arm (27). MAP2c not only has a similar molecular weight to 7, but also possesses a nearly identical microtubule-binding domain (12). It might therefore be predicted that much of MAP2c interacts with microtubules and that a small projection domain interacts with the regulatory subunit of the CaM-dependent kinase II (14). These biophysical data of high molecular weight MAP2 stand somewhat in contrast to both rotary shadowing and electron microscopic data which suggest that the arm of high molecular weight MAP2 is an extended semirigid rod-like molecule (7, 30) which might be folded back on itself (31). Perhaps the answer is somewhere in between since our computer-generated secondary structure predictions suggest that although a portion of MAP2 exists as a random coil, one cannot fail to notice that beginning near the MAP2c junction and extending toward the amino terminus is a very significant stretch of helices separated by short turns. This motif could theoretically permit MAP2 to exist in an extended flexible conformation yet perform a structural role in the neuronal cytoskeleton.

Binding Motifs and MAP2 Phosphorylation—Post-transla-

* B. Schwanke, B. Schultz, and C. C. Garner, unpublished observations.
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togenesis (42). Finally, Viereck et al. (43) have shown that MAP2c is present in regions of the adult brain which are constantly undergoing regeneration. This suggests that the ratio of MAP2b to MAP2c in developing neurons helps to determine the extent of cross-linking and thus the plasticity of the dendritic cytoskeleton. This hypothesis, of course, depends on the yet unproven co-distribution of MAP2b and MAP2c.

Acknowledgments—We would like to thank Abby Garner and Birgit Schwanke for their technical assistance and Gunter Schuetz at the Deutscher Krebs Forschung Zentrum, Heidelburg, for the rat genomic library. We would also like to thank Scott Daniels, of Milligen, Burlington, MA, for the synthesis and purification of the cyclized peptide, as well as Eckart Gundelfinger and Arnd Baumann for critical reading of the manuscript.

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Molecular structure of microtubule-associated protein 2b and 2c from rat brain.
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J. Biol. Chem. 1990, 265:19679-19684.

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