Molecular Characteristics and Interactions of the Intermediate Filament Protein Synemin

INTERACTIONS WITH α-ACTININ MAY ANCHOR SYNEMIN-CONTAINING HETEROFILAMENTS

(Received for publication, June 14, 1999, and in revised form, July 30, 1999)

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Synemin is a cytoskeletal protein originally identified as an intermediate filament (IF)-associated protein because of its colocalization and copurification with the IF proteins desmin and vimentin in muscle cells. Our sequencing studies have shown that synemin is an unusually large member (1,604 residues, 182,187 Da) of the IF protein superfamily, with the majority of the molecule consisting of a long C-terminal tail domain. Molecular interaction studies demonstrate that purified synemin interacts with desmin, the major IF protein in mature muscle cells, and with α-actinin, an integral myofibrillar Z-line protein. Furthermore, expressed synemin rod and tail domains interact, respectively, with desmin and α-actinin. Analysis of endogenous protein expression in SW13 clonal lines reveals that synemin is coexpressed and colocalized with vimentin IFs in SW13.C1 vim+ cells but is absent in SW13.C2 vim− cells. Transfection studies indicate that synemin requires the presence of another IF protein, such as vimentin, in order to assemble into IFs. Taken in toto, our results suggest synemin functions as a component of heteropolymorphic IFs and plays an important cytoskeletal cross-linking role by linking these IFs to other components of the cytoskeleton. Synemin in striated muscle cells may enable these heterofilaments to help link Z-lines of adjacent myofibrils and, thereby, play an important role in cytoskeletal integrity.

Intermediate filaments (IFs), along with actin-containing microfilaments and tubulin-containing microtubules, are one of the three major classes of cytoskeletal filaments in multicellular animals (1–4). The IFs, which are considered to play an important role in structure and mechanical integration of cellular space (5, 6), are composed of cell type-specific proteins that have been divided into classes based upon sequence comparisons (1, 3, 7). The members of this protein superfamily have within their sequence a conserved rod domain, which promotes coiled-coil interactions between two individual IF proteins and formation of an IF protein dimer, the first step in assembly of the ~10 nm diameter IFs (1, 3, 8–11). Flanking the rod domain are N-terminal head and C-terminal tail domains that vary considerably in size and sequence among the IF protein classes (1, 3, 4). Most IF proteins are grouped into five major classes or types (I–V) based upon sequence analysis (1–4). Some classes of IF proteins, such as the type I and II keratins (3) and the type IV neurofilament proteins (12, 13), are known to form obligate heteropolymers in vivo, resulting in IFs that consist of at least two different IF proteins. In contrast, IFs containing type III proteins, such as desmin or vimentin, are often considered homopolymeric IFs (3, 14) because each of these individually purified proteins readily assemble into synthetic IFs in vitro (8, 15–17).

The type III IF proteins vimentin and desmin are the major IF proteins of developing and mature striated muscle cells, respectively (18, 19). Synemin and paranemin, a pair of relatively high molecular weight proteins identified in the early 1980s, were initially described as IF-associated proteins because they copurified in the initial purification steps with desmin and vimentin and colocalized with them in muscle cells (2, 20–24). Recent cloning and sequencing studies in our laboratory, however, demonstrate that both synemin and paranemin contain the ~310-amino acid rod domain characteristic of IF proteins and, therefore, are members of the IF protein superfamily (25, 26). Those results, along with their colocalization (20, 22, 26, 27), suggest synemin and paranemin may form heteropolymeric IFs with the type III proteins desmin and/or vimentin in vivo (26, 27).

Our hypothesis is that synemin acts as a component of heteropolymeric IFs with vimentin and/or desmin and helps attach these IFs to other cytoskeletal structures. Based upon localization of IFs at the periphery of, and between, Z-lines of adjacent myofibrils (19, 28, 29), synemin-containing heteropolymeric IFs may help link adjacent myofibrils in striated muscle cells. In this paper, we describe the complete sequence of synemin, which establishes it as a unique IF protein with a long C-terminal extension, which is not readily grouped with any of the well established IF protein types. Transfection of full-length synemin into SW13 clonal lines demonstrates that synemin requires another IF protein for assembly into IFs. We demonstrate specific molecular interactions between synemin...
and desmin, the major IF protein present in most mature muscle cells (18, 19), and between the large tail domain of synemin and α-actinin, an integral protein of myofibrillar Z-lines (30) and costameres (31) of striated muscle cells, and of adhesion plaques of many other cell types (32). In toto, the studies herein help establish synemin as an important member of the IF protein superfamily and one that likely functions as a component of heteropolymeric IFs that can interact with α-actinin and, thereby, enable IFs to link other components of the cytoskeleton.

**Experimental Procedures**

**Cloning and Sequencing of Avian Synemin cDNA**—Initial cloning studies on synemin from our laboratory (25) described only sequence of the rod domain portion of synemin. Additional clones encoding parts of the full-length synemin cDNA were retrieved from the same λgt11 library, prepared from adult chicken gizzard, by hybridization screening. Sequencing of the entire length of both strands of clones 47, 108, 135 and 244, and multiple internal sites of all other clones shown in Fig. 1, was done on Applied Biosystems 373 and 377 sequencers at the Iowa State University Sequencing and Synthesis Facility. Confirmation of the 5′ end of the sequence was done by 5′-rapid amplification of cDNA ends (RACE) kit from Life Technologies, Inc., by using a primer (nucleotides 144–162) from the 5′ end of clone 108. The longest rapid amplification of cDNA ends clones produced start at the same nucleotide as the 244 clone. Computer analysis of the synemin cDNA sequence was carried out by using version 10 of the Wisconsin Package, Genetics Computer Group (GCC), Madison, WI, and the NCBI BLAST server (34).

**Antibodies**—Synemin polyclonal antibodies (pAb) 2856 were produced in rabbits injected with native purified protein essentially as described (35). The pAbs were characterized by Western blotting, and they labeled only the 230-kDa synemin band present in fresh, avian whole muscle homogenates. Additionally, these antibodies labeled purified samples of both the expressed rod and C-terminal tail domains of synemin. Aliquots of these antibodies also were affinity purified by utilizing a column of purified, intact synemin coupled to CNBr-activated Sepharose 4B (Sigma). Vimentin monoclonal antibody (mAb) AMP-17b (developed by Dr. A. B. Fulton) was obtained from the Developmental Studies Hybridoma Bank.

**Immunocytocchemistry and Western Blotting**—Immunocytocchemistry studies with SW13.C1 vim+ and SW13.C2 vim− cells were done similarly to those described in Hemken et al. (26) but utilized synemin pAb 2856. For Western blotting, cell lysates of SW13.C1 vim+ and SW13.C2 vim− cells were separated into supernatant and pellet fractions to concentrate the cytoskeletal proteins in the pellets by the method of Athlan et al. (36). The resulting samples were analyzed by standard procedures (37), using enhanced chemiluminescence (ECL) and blot striping according to the manufacturer’s procedure described in the ECL Western blotting protocols guidebook (Amersham Pharmacia Biotech).

**Northern Blotting**—Total RNA was prepared from SW13.C1 vim+ and SW13.C2 vim− cells by the standard guanidine isothiocyanate method (38), and blasts were probed with avian cDNA probes corresponding to either the synemin rod or tail domain by using the Gene-Images kit (Amerham Pharmacia Biotech). The kit instructions were followed, except that the gel transfer was carried out by using 50 mM NaOH as the transfer fluid, and the hybridization and final stringency washes were done at 47 °C.

**Cell Transfection Assays**—Studies were carried out essentially as described (26), with the following changes. Full-length synemin cDNA was assembled from overlapping clones and inserted into the pCDNA3 eukaryotic expression vector (Invitrogen). The cDNA construct was transfected into SW13.C2 vim− cells with FuGENE 6 reagent (Roche Molecular Biochemicals), utilizing an empirically determined ratio of 3 μl of transfection reagent to 1 μg of DNA for a 60-mm diameter dish. Proteins were visualized approximately 40 h after transfection by immunocytocchemistry.

**Protein Purification**—All proteins purified from tissue were prepared from adult turkey gizzards quick frozen immediately postmortem to minimize proteolysis. Synemin, in particular, is highly susceptible to proteolytic degradation (27). Intact synemin (27), desmin (15), and α-actinin (39) were purified by standard methods. The rod (nucleotides 138–1047) and tail (nucleotides 1048–4917) domains of synemin were produced by bacterial expression using pProEX HT vectors (Life Technologies, Inc.). The rod domain was expressed in Escherichia coli XL-1

![FIG. 1. Alignment of synemin cDNA clones](http://www.jbc.org/)
Molecular Characterization and Interactions of Synemin

FIG. 2. Amino acid sequence of avian muscle synemin deduced from its cDNA sequence. Amino acids are designated by the one-letter code. **Bold type** denotes the extent of the conserved rod domain typical of IF proteins. The extent of each of the subdomains within the rod domain is as follows: 1A, residues 11–46; 1B, residues 47–57; 1C, residues 58–152; 2A, residues 153–166; 2B, residues 167–206; 3A, residues 207–217; and 3B, residues 218–314. The “intermediate filament signature,” identified with the GCG program Motifs, is underlined. The nucleic acid sequence of synemin is available from GenBank under accession number U28143.

FIG. 3. Western blot analysis of endogenous expression of synemin and vimentin in SW13 cells. Panels A and B depict the identical blot of fractionated cell lysates, with A probed with synemin pAb 2556, and B stripped and re-probed with vimentin mAb AMF-17b. Lane 1, avian gizzard homogenate (a stored sample exhibiting synemin and its proteolytic degradation products in panel A (27), and the small amount of expected vimentin in panel B); lane 2, SW13.C1vim+ supernatant; lane 3, SW13.C1vim+ + pellet; lane 4, SW13.C2vim− supernatant; and lane 5, SW13.C2vim− + pellet. The approximate migration distances of filamin (250 kDa), myosin heavy chain (205 kDa), α-actinin (100 kDa), desmin (53 kDa), and actin (42 kDa) from the gizzard homogenate are indicated.

for a protein with a predicted molecular mass of 182,187 Da. The sequence mass is smaller than that estimated (230 kDa) for synemin by SDS-PAGE (20, 27). This size difference may be explained, as it was for the IF protein paranemin (26), by the absence of the cytoplasmic IF protein vimentin. We first characterized the cell line before we used it for the transfection studies. Western blot analysis of lysates of these clonal lines (Fig. 3) showed, as expected, that synemin was absent in the SW13.C2 vim− cells (Fig. 3, panel A, lanes 4 and 5) but, surprisingly, that synemin was already present in the cytoskeletal protein-containing pellet fraction of SW13.C1 vim+ cells (Fig. 3, panel B, lane 3). Northern blot analysis showed that the mRNA for synemin (~9 kb) was present in SW13.C1 vim+ cells but absent in the SW13.C2 vim− cells. Thus, synemin has the same pattern of transcription as previously shown for the mRNA of vimentin in these clonal lines (42). Double label immunofluorescence of SW13.C1 vim+ cells, utilizing the same antibodies as for the Western blotting experiments, also show that they express synemin as a component of their vimentin-containing IF network (Fig. 4, panel B, lane 3). Northern blot analysis showed that the mRNA for synemin (~9 kb) was present in SW13.C1 vim+ cells but absent in the SW13.C2 vim− cells. Thus, synemin has the same pattern of transcription as previously shown for the mRNA of vimentin in these clonal lines (42). Double label immunofluorescence of SW13.C1 vim+ cells, utilizing the same antibodies as for the Western blotting experiments, also show that they express synemin as a component of their vimentin-containing IF network (Fig. 4, panels A and B). And, consistent with the Western and Northern blot analyses, immunofluorescence labeling of the SW13.C2 vim− cells, known to lack an endogenous vimentin IF network, with synemin pAbs showed no labeling of synemin (Fig. 4, panels C and D).

Transfection of SW13.C2 vim− cells with full-length synemin cDNA resulted in cells that contain punctate aggregates when observed by immunofluorescence (Fig. 4, panels E and F). The punctate aggregates were similar to those seen for paranemin expressed in the same vimentin-negative cell line (26) and for assembly-deficient mutants of desmin in other cells lacking IFs (44). These results suggest that synemin cannot form an IF network without another IF protein, such as vimentin, present.

**Protein Interaction Studies**—Experiments were conducted to characterize interactions between synemin and desmin. Analysis of the interactions by cosedimentation of purified desmin and purified synemin is shown in Fig. 5. Under non-IF forming conditions (Fig. 5A), neither desmin alone (panel 1) nor synemin alone (panel 2) sedimented. However, even under these conditions that are unfavorable for IF formation, synemin and desmin interact as shown by the presence of a significant portion of both proteins in the pellet fraction (panel 3). Under conditions favorable for IF formation (Fig. 5B), desmin by itself line was utilized. This cell line has been separated into specific clonal lines, including the SW13.C1 vim−, which has an endogenous vimentin IF network, and the SW13.C2 vim−, which lacks any cytoplasmic IFs (42). These clonal lines have been used in several studies (e.g., Refs. 26 and 43) to characterize assembly of transfected IF proteins into IFs in the presence and absence of the cytoplasmic IF protein vimentin. We first characterized the cell line before we used it for the transfection studies. Western blot analysis of lysates of these clonal lines (Fig. 3) showed, as expected, that synemin was absent in the SW13.C2 vim− cells (Fig. 3, panel A, lanes 4 and 5) but, surprisingly, that synemin was already present in the cytoskeletal protein-containing pellet fraction of SW13.C1 vim+ cells (Fig. 3, panel B, lane 3). Northern blot analysis showed that the mRNA for synemin (~9 kb) was present in SW13.C1 vim+ cells but absent in the SW13.C2 vim− cells. Thus, synemin has the same pattern of transcription as previously shown for the mRNA of vimentin in these clonal lines (42). Double label immunofluorescence of SW13.C1 vim+ cells, utilizing the same antibodies as for the Western blotting experiments, also show that they express synemin as a component of their vimentin-containing IF network (Fig. 4, panels A and B). And, consistent with the Western and Northern blot analyses, immunofluorescence labeling of the SW13.C2 vim− cells, known to lack an endogenous vimentin IF network, with synemin pAbs showed no labeling of synemin (Fig. 4, panels C and D).

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FIG. 4. Immunofluorescence localization of synemin and vimentin in SW13 cells. Panels on the left (A, C, and E) depict immunofluorescence labeling with synemin pAb 2856. Panels on the right (B, D, and F) depict fluorescence labeling with vimentin mAb AMF-17b. Panels A and B show endogenous synemin/vimentin expression in a filamentous pattern typical of IFs in the SW13 C1 vim + cells. Panels C and D show the lack of endogenous synemin/vimentin expression in the SW13.C2 vim− cells. Note that synemin colocalizes with vimentin in SW13.C1 vim + cells (panels A and B) but is absent from SW13.C2 vim− cells as is vimentin (panels C and D). Panels E and F show immunofluorescent labeling of SW13.C2 vim− cells after transfection with full-length synemin cDNA. Note that the synemin expressed in the SW13.C2 vim− cells appears in a non-filamentous, punctate pattern when vimentin is absent (panel E). Bar, 10 μm for A–F.

FIG. 5. Cosedimentation of purified synemin with purified desmin. A, SDS-PAGE analysis of results from mixing purified synemin with desmin in non-IF forming conditions (10 mM Tris-HCl, pH 8.5). B, SDS-PAGE analysis of results from mixing purified synemin with desmin in non-IF forming conditions and then adjusting the buffer conditions to induce filament formation (100 mM NaCl, 1 mM MgCl2, pH 7.0) before centrifugation. C, SDS-PAGE analysis of results from mixing purified synemin with preformed desmin filaments. For A–C, panel 1 is desmin alone, without synemin; panel 2 is synemin alone, without desmin; and panel 3 is desmin and synemin mixed; S, supernatant; P, pellet. BSA was added to all samples to show that virtually no exogenous (unbound) protein (e.g., synemin rod) was simply trapped within the volume of the pellet(s).

IF-forming conditions, nearly all of the synemin sedimented with the desmin (panel 3). In contrast to the results shown in Fig. 5B (panel 3), much less synemin interacted with “preformed” desmin filaments (Fig. 5C, panel 3). Overall, these results show that synemin and desmin interact (Fig. 5A, B and C, panel 3) and suggest that synemin has greater access and/or affinity for binding free desmin molecules available before and during filament formation (Fig. 5B, panel 9), in comparison to binding to desmin already incorporated into IFs (Fig. 5C, panel 3).

Interaction studies of the bacterially expressed rod domain of synemin with purified desmin (pre-mixed at low ionic strength and then the buffer adjusted to favor IF-forming conditions) are shown in Fig. 6. Whereas desmin by itself is sedimented (panel 1), only about half of the synemin rod domain by itself is sedimented (panel 2). However, essentially all of the synemin rod domain is sedimented in the presence of desmin (panel 3), confirming the rod domain of synemin as a major site of interaction with desmin. The lack of BSA in the pellets (Figs. 5 and 6, all panels) in the cosedimentation assays indicates that the increased amount of synemin (Fig. 5) or of synemin rod (Fig. 6) in the pellets is due to a specific interaction with desmin.

Blot overlay analyses utilizing purified desmin, α-actinin, and synemin, as well as bacterially expressed rod and tail domains of the synemin molecule, were done to identify specific protein interactions. α-Actinin was selected as a potential interaction partner both because it is a major integral Z-line protein (30) and because the synemin-containing IFs very closely encircle the Z-lines in muscle.2 As shown in the control lacking any probe protein in the overlay (Fig. 7, panel B) the synemin pAb 2856 labeled only the purified synemin (lane 4) and the synemin in the gizzard homogenate (lane 1) but did not label either desmin (lanes 1 or 2) or α-actinin (lanes 1 or 3). As shown in Fig. 7 (panel C), probing blots of purified desmin and α-actinin with purified intact synemin reveals significant interactions of synemin with both desmin (lanes 1 and 2) and α-actinin (lane 3). If overloaded samples of gizzard homogenate were run in lane 1, an interaction between synemin and the α-actinin in the gizzard homogenate also was detected.3 As shown in Fig. 7 (panel D), probing the blots with the bacterially expressed synemin rod domain reveals that it interacts with desmin (lanes 1 and 2) but not with α-actinin (lane 3). As shown in Fig. 7 (panel E), probing blots with the bacterially expressed synemin tail domain demonstrates it interacts with both desmin (lanes 1 and 2) and α-actinin (lane 3). Specificity of the

2 M. M. Bilak and R. M. Robson, unpublished observations.
3 S. W. Sernett and R. M. Robson, unpublished observations.
The cDNA sequence identities of the avian synemin cDNA with the human EST (aligns with the last two-thirds of the synemin rod domain sequence with 58% sequence identity) and with human brain cDNA clone AB002351 (51) (aligns with the synemin sequence starting from within subdomain 2B near the end of the rod domain to the middle of the 3'-UTR with 48% sequence identity), which in turn are 99% identical to each other at the nucleotide level, suggest that human synemin has been partially sequenced. If this does represent human synemin sequence, it exhibits considerable divergence from the avian synemin sequence for much of the length of the sequence currently available. When the amino acid sequences predicted from these cDNA sequences are compared, avian synemin and the human AB002351 clone exhibit only 41% homology (33% amino acid identity). However, it is striking that the extreme C-terminal end (50 residues) of synemin is almost identical to the protein predicted from the AB002351 clone, including the same stop codon position. At a minimum, if this cDNA from human brain does not code for synemin, there exists a protein in human brain cells that exhibits significant homology to avian synemin. Additionally, our identification of endogenous synemin in SW13.C1 vim+ cells, a cell line derived from a human adrenal cortex adenocarcinoma, indicates the existence of human synemin. Western blotting of this endogenous human synemin indicated a molecular mass of approximately 225 kDa, slightly lower than observed for avian muscle synemin, but essentially identical to what we identified (225 kDa) in porcine muscle (27). Additionally, although the specific, full-length human synemin cDNA has not yet been sequenced, the approximate size of the human mRNA (~9.0 kb) we described herein is very close to the 8.4 kb identified for synemin in avian tissue (25).

With increasing interest in so-called 3'-UTR “zip codes,” which have been shown to play a role in the localization of the mRNAs of other cytoskeletal proteins including specific actin isoforms and even the IF protein vimentin (52, 53), it is possible that such a sequence may exist within the notably large synemin 3'-UTR. Unfortunately, in most cases no true sequence homology domains exist for the zip codes, making the identification of such a sequence difficult (53).

It has been speculated since its discovery (20) that synemin functions within cells in close association with the IF proteins desmin and/or vimentin (20, 22, 25, 27, 54). That the sequence of synemin demonstrates it also is an IF protein strongly suggests that interactions between synemin and the type III IF proteins desmin and/or vimentin will include those of their rod domains. Our transfection studies in SW13 cells explored...
whether synemin, by itself, could assemble into IFs. Interactions that have been identified between two members of the IF protein superfAMILY (e.g. type I and II keratins, desmin and vimentin, NF-L with NF-M and/or NF-H) are known to involve their rod domains (3, 47). The colocalization of synemin and vimentin in the IF network in the SW13.C1 vim+ cells and the lack of IF formation in the SW13.C2 vim− cells lacking vimentin, but expressing synemin from transfection, suggest that synemin forms heteropolymeric IFs along with another IF protein but does not form IFs by itself. These in vivo findings are in concert with the in vitro cosedimentation results, which showed that synemin, by itself in IF-forming conditions, remained in the supernatant (Fig. 5B, panel 2) but was sedimented maximally in the presence of desmin when the two proteins were first mixed under conditions unfavorable for IF formation and then converted to conditions favoring filament formation prior to centrifugation (Fig. 5B, panel 3). That the assembly of synemin into IFs depends on the presence of another IF protein also provides additional insight into an early report (55), which showed that the rate of vimentin filament assembly limited the rate at which synemin became part of the insoluble fraction isolated from cell lysates (i.e. synemin required the presence of another IF protein for incorporation into IFs).

The results of our cosedimentation studies demonstrated a specific interaction between intact synemin and desmin (Fig. 5) and between the synemin rod domain and desmin (Fig. 6), especially under filament-forming conditions. Those interactions were verified by using a different technique, namely the blot overlay studies (Fig. 7). These interactions are consistent with studies demonstrating colocalization and initial copurification of synemin with desmin from adult avian (27, 56) and mammalian (27) muscle tissue. The blot overlay studies also revealed an interaction between the synemin tail domain and desmin (Fig. 7), which may reflect the staggered array of IF proteins present in the assembly intermediates involved in IF assembly (3, 8, 11, 17). Furthermore, specific sites within the cytoskeletal structures, containing IFs may serve to link directly these particular IFs to other cytoskeletal structures, containing IFs and between the synemin tail domain and desmin (Fig. 5), and between the synemin rod domain and desmin and between the synemin tail domain and the actin-binding protein α-actinin support our working hypothesis for the role of synemin. As a component of heteropolymeric IFs, synemin would be able to link directly the IFs to α-actinin-containing structures. In the case of striated muscle cells, synemin may play an important role in directly linking the heteropolymeric IFs to α-actinin within the myofibrillar Z-lines and costameres. Thus, synemin-containing IFs could firmly link all adjacent myofibrils within the cell and the peripheral layer of myofibrils to the muscle cell membrane. This linkage may help maintain overall cytoskeletal integrity and contractile function.

Acknowledgments—We thank Stephan Bilak, Dr. Marvin H. Stromer, and Stephanie Seiler, Iowa State University, for performing initial interaction studies on synemin, assistance with fluorescence microscopy, and useful discussions on IF proteins, respectively. The Developmental Studies Hybridoma Bank is maintained by the Department of Pharmacology and Molecular Sciences, The Johns Hopkins University School of Medicine, Baltimore, MD 21205, and the Department of Biological Sciences, University of Iowa, Iowa City, IA 52242, under NICHD Grant NO1-HD-2–3144 from the National Institutes of Health.
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doi: 10.1074/jbc.274.41.29493

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