Two Caenorhabditis elegans Actin Depolymerizing Factor/Cofilin Proteins, Encoded by the unc-60 Gene, Differentially Regulate Actin Filament Dynamics*

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The Caenorhabditis elegans unc-60 gene encodes two actin depolymerizing factor/cofilin proteins which are implicated in the regulation of actin filament assembly in body wall muscle. We examined the interaction of recombinant UNC-60A and B proteins with actin and found that they differentially regulate actin filament dynamics. Co-pelleting assays with F-actin showed that UNC-60A depolymerized but did not remain bound to F-actin, whereas UNC-60B bound to but did not depolymerize F-actin. In the pH range of 6.8–8.0, the apparent activities of UNC-60A and B did not change although UNC-60A showed greater actin-depolymerizing activity at higher pH. These activities were further confirmed by a light scattering assay and electron microscopy. The effects of these proteins on actin polymerization were quite different. UNC-60A inhibited polymerization in a concentration-dependent manner. On the other hand, UNC-60B strongly inhibited the nucleation process but accelerated the following elongation step. However, an excess amount of UNC-60B increased the amount of unpolymerized actin. These results indicate that UNC-60A depolymerizes actin filaments and inhibits actin polymerization, whereas UNC-60B strongly binds to F-actin without depolymerizing it and, through binding to G-actin, changes the rate of actin polymerization depending on the UNC-60B:actin ratio. These data suggest that the two UNC-60 isoforms play differential roles in regulating actin filament dynamics in vivo.

Myofibrils are highly differentiated forms of actin cytoskeleton that are specialized for muscle contraction, but the mechanisms by which these complex and precise structures are assembled and maintained are largely unknown. Actin, a major component of thin filaments, has an inherent tendency to polymerize into filaments in vitro. However, the assembly of actin in developing muscle is regulated, and consequently, about 40% of actin is present in a monomeric form (1). In embryonic chicken skeletal muscle, proteins that bind to G-actin to prevent them from polymerization have been identified as profilin (2), actin depolymerizing factor (ADF)1 (3), and cofilin (4).

Quantitative analysis has shown that the concentrations of these three proteins are sufficient for sequestering most of G-actin at a late stage of embryonic muscle (5), suggesting that they are responsible for regulating actin filament assembly. ADF and cofilin are highly conserved proteins, are members of an ADF/cofilin family having 25–71% homology, and are found in diverse organisms. ADF/cofilin binds to both G- and F-actin at a stoichiometry of 1:1 and regulates the rate of actin polymerization (reviewed in Ref. 6). Recently, ADF/cofilin has been shown to affect the on/off rates at both ends of F-actin, which results in the enhancement of treadmilling (7). This function is necessary for the actin-based motility of Listeria monocytogenes (7, 8) and for actin turnover in cortical actin patches in yeast (9).

A muscle-specific function for ADF/cofilin has been suggested by two examples. These are a mammalian muscle-specific cofilin (M-cofilin) (10) and two ADF/cofilin homologues encoded by the Caenorhabditis elegans unc-60 gene (11). Mammalian M-cofilin is predominantly expressed in skeletal and cardiac muscles (10), but its exact function is unknown. Mutations in the unc-60 gene result in slow moving or paralyzed nematodes (11–13). By electron microscopy, unc-60 mutant muscle has large accumulations of thin filaments especially at the ends of muscle cells but only a few thin filaments scattered among thick filaments that are present in normal numbers and roughly organized into A-bands (12). Thus, unc-60 is required for proper positioning and the correct number of thin filaments in nematode striated muscle. The unc-60 gene has been shown to encode two transcripts, sharing only a single exon encoding the initiator methionine, and two homologous proteins of the ADF/cofilin family (11). These proteins, called UNC-60A and UNC-60B, are 165 and 152 amino acids long, respectively, and are 36% identical and 72% similar. Biochemical studies on members of the ADF/cofilin family in other organisms suggest that the UNC-60 proteins regulate actin polymerization. But, analysis of the primary structures of the UNC-60 isoforms does not allow us to predict how their biochemical properties might be different. We need to know the precise biochemical properties of the UNC-60 proteins to understand the role of unc-60 in muscle development. To address this question, we studied the biochemical characteristics of two UNC-60 proteins, and found that they regulate actin filament dynamics in different manners. The results suggest that two UNC-60 proteins have physiologically distinct functions.

EXPERIMENTAL PROCEDURES

Construction of Expression Vectors for UNC-60A and B—cDNAs for UNC-60A and UNC-60B were amplified from a C. elegans cDNA library (kindly provided by Dr. R. Barstead, Oklahoma Medical Research Foundation) by polymerase chain reaction with Pfu DNA polymerase (Stratagene, Inc.). Forward and reverse primers for UNC-60A were 5’-GATCCATATGAGTCCGGTGTATCTAGTGATCTCC and 5’-GATTGGATCATCGTGATCTATCGTGATCTCC. Forward and reverse primers for UNC-60B were 5’-CTAGTGATCTCC. Forward and reverse primers for UNC-60B were 5’-GATCCATATGAGTCCGGTGTATCTAGTGATCTCC. Forward and reverse primers for UNC-60B were 5’-GATCCATATGAGTCCGGTGTATCTAGTGATCTCC. Forward and reverse primers for UNC-60B were 5’-GATCCATATGAGTCCGGTGTATCTAGTGATCTCC.

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1 The abbreviations used are: ADF, actin depolymerizing factor; EDC, 1-ethyl-3-(3-dimethylamino)propyl)carbodiimide; Pn, inorganic phosphate; PAGE, polyacrylamide gel electrophoresis.
60B were 5'-GATCCCATGCTCTCGGAGTCAAGTGG and 5'-CTA-GGGATCCCGCATGATTTTGGACATC. The amplified products for A and B were respectively digested by NdeI-BamHI and NcoI-BamHI, at the sites introduced by the primers, and then cloned into pET-3a and pET-3d (Novagen). The sequences of the inserts were verified by DNA sequencing not to contain any polymersac chain reaction-induced errors.

Preparation of Recombinant UNC-60 Proteins—Basically, UNC-60A and UNC-60B were produced and purified by the same method. The Escherichia coli strain BL21 (DE3) pLysS carrying an expression vector was grown in LB medium containing 50 μg/ml ampicillin at 37 °C until the A600 reached 0.6. Protein expression was induced by adding 0.4 mM isopropyl β-D-thiogalactopyranoside for 2 h at 37 °C. The cells were harvested by centrifugation and disrupted by a French pressure cell in a buffer containing 0.1 M NaCl, 1 mM EDTA, 10 mM HEPES-NaOH, pH 7.5, and then monitoring the light scattering at an angle of 90° and a wavelength of 500 nm was measured with a fluorescence spectrophotometer (Perkin-Elmer LS50B). Samples were negatively stained with 1% uranyl acetate aqueous solution on carbon-supported Formvar-coated grids and observed with a JEOL 100CX electron microscope at an accelerating voltage of 80 kV.

Assay for Actin Polymerization—G-actin was prepared from rabbit muscle acetone powder as described (4). The time course of actin polymerization was monitored by measuring the absorbance at 237 nm; increased absorbance reflects the G- to F-actin transformation (15, 16). The assay was performed at 25 °C with an Ultrospec 3000 spectrophotometer (Pharmacia Biotech Inc.) equipped with a water-heated 6-cell changer. G-actin (5 μM) was mixed with various concentrations of UNC-60A or B proteins in a buffer containing 0.2 mM ATP, 20 μM CaCl2, 0.2 mM dithiothreitol, 2 mM Tris-HCl, pH 8.0, and incubated for 15 min. The polymerization was started by adding salt and buffer to a final concentration of 0.1 M KCl, 2 mM MgCl2, 1 mM EGTA, 20 mM HEPES-NaOH, pH 7.5, and then monitoring the A237.

RESULTS

Effects of UNC-60 Proteins on F-actin—Recombinant UNC-60A and B proteins without any extra sequences were bacterially produced, purified (Fig. 1), and used in the following experiments. First, we found that their effects on F-actin were quite different. The actin-binding activities of UNC-60A and B were examined by a co-pelleting assay with preassembled F-actin (Fig. 2). In the presence of UNC-60A, the amount of unpolymerized actin was increased in the supernatant, whereas UNC-60A did not co-sediment with F-actin (Fig. 2A), indicating that UNC-60A primarily depolymerized actin filaments and bound to G-actin. Nearly complete depolymerization was observed when a two molar excess or more of UNC-60A was added (Fig. 2A, graph). In contrast, UNC-60B co-sedimented with F-actin but did not increase the amount of unpolymerized actin (Fig. 2B), showing that UNC-60B bound to F-actin but did not have actin-depolymerizing activity. UNC-60B alone precipitated only at a negligible amount (less than

FIG. 1. Purified recombinant UNC-60A and B proteins and an alignment of their sequences. Bacterially expressed UNC-60A (A) and UNC-60B (B) were purified as described under "Experimental Procedures," and 5 μg of each protein was separated by SDS-PAGE with a 15% gel. Molecular mass markers in kDa are indicated on the left. Below is an alignment of UNC-60A and B amino acid sequences performed by ALIGN at the GeneStream Search network server (CRBM, Montpellier, France). Double and single dots indicate identical and similar amino acids, respectively. They are 35.5% identical and 72.3% similar. The underline emphasizes the eight extra residues found in UNC-60A that, based on the yeast cofilin structure, are likely to reside in a β-sheet exposed on the surface of the protein.
3% of total protein, data not shown). The binding of UNC-60B to F-actin was saturated at a molar ratio of 1:2:1 (Fig. 2B), suggesting that the stoichiometry of the binding was 1 to 1. ADF/cofilins in vertebrates, yeast, and plants have been shown to have a pH-dependent F-actin-binding/actin-depolymerizing activity; they bind to F-actin at pH 6.5–7.1 and depolymerize actin filaments at pH 7.3–8.3. Therefore, we performed the co-pelleting assay at pH 6.8, 7.5, and 8.0. However, the apparent activities of UNC-60A and B were not changed under the conditions examined, although the actin-depolymerizing activity of UNC-60A was stronger at higher pH (data not shown).

The effects of UNC-60 proteins on F-actin were kinetically measured by light scattering (Fig. 3). Fig. 3A shows the stability of the F-actin polymers without the addition of UNC-60 proteins during the course of these experiments. As shown in Fig. 3B, UNC-60A initially increased and then decreased the scattering intensity. This suggests a transient association of UNC-60A with F-actin which is followed by rapid depolymerization of F-actin, so that its F-actin binding was not able to be detected by the pelleting assay. As shown in Fig. 3C, UNC-60B, in contrast, increased the scattering intensity in a biphasic manner. This confirms the results of the pelleting assay in which UNC-60B binds to, but does not depolymerize F-actin. The biphasic increase in light scattering suggests that binding of UNC-60B to actin is cooperative.

The effects of UNC-60 proteins on actin filaments were further examined by electron microscopy (Fig. 4). In the presence of UNC-60A, only a few long actin filaments were observed, and short filaments were frequently found (Fig. 4B), indicating that F-actin was depolymerized by UNC-60A. In contrast, UNC-60B maintained long actin filaments and no obvious effect was observed at this resolution (Fig. 4C). Nevertheless, on closer examination, in the presence of UNC-60B, actin filaments seemed to be more straight rather than the kinky shape that was often found with F-actin alone. This suggests that UNC-60B changes the structure of F-actin similarly to that reported for human cofilin (19).

The binding of the UNC-60 proteins to the actin monomer was estimated to be at a 1:1 ratio by a cross-linking assay using a zero-length cross-linker, EDC (Fig. 5). EDC has been shown previously to cross-link actin and ADF/cofilin family proteins but not to cross-link actin monomers within actin polymers (3, 4, 20, 22). In the presence of the cross-linker in mixtures of actin and UNC-60A or UNC-60B, new bands of approximately 60 kDa appeared (Fig. 5, lanes 3 and 6, arrow) on the gel, which are equivalent to the sum of one actin (42 kDa) molecule and one UNC-60A (20 kDa) or B (18 kDa) molecule. These bands did not appear when the cross-linker was incubated with actin or UNC-60 proteins alone (Fig. 5, lanes 1, 2, and 5), or actin and UNC-60 proteins were incubated without the cross-linker (Fig. 5, lanes 4 and 7), indicating that both UNC-60A and UNC-60B bind to actin at a molar ratio of 1:1. These results are consistent with the previous studies on other ADF/cofilin family members (3, 4, 20–22).

Effects of Inorganic Phosphate on the Interactions of UNC-60 Proteins with Actin—Both the actin-depolymerizing activity of UNC-60A and the F-actin binding activity of UNC-60B were inhibited by inorganic phosphate (Pi) (Fig. 6). Addition of Pi to mixtures of F-actin and UNC-60A decreased the amount of actin in the supernatant (Fig. 6A). Similarly, in the presence of Pi, the amount of UNC-60B that was pelletable with actin was decreased (Fig. 6B). As a control, increasing the potassium concentration by adding potassium chloride did not affect the activities of the UNC-60 proteins (data not shown). Pi has been shown to bind to F-actin with an affinity of several millimolar (23) and create the state of ADP-Pi-actin which is the intermediate in the hydrolysis of ATP into ADP. Therefore, these results suggest that both UNC-60A and B preferentially bind to ADP-bound actin within the filaments in agreement with the properties of actophorin and plant ADF (7, 22).

Effects of UNC-60 Proteins on Actin Polymerization—UNC-60A and B affect the kinetics of actin polymerization in different manners (Fig. 7). Fig. 7A shows that, in the presence of
UNC-60A, the rate of polymerization was slowed and the amount of polymerized actin was decreased in a concentration-dependent manner. The early phase of actin polymerization was strongly inhibited by equal molar or 2 molar excess of UNC-60A, suggesting that UNC-60A bound to G-actin, so that it inhibited the nucleation step of actin polymerization and sequestered actin monomer to prevent polymerization. However, two molar excess of UNC-60A transiently enhanced the polymerization rate (from 1,000 to 2,000 s), which may be due to a weak filament severing activity similar to that reported for actophorin (22) and ADF (24, 25).

UNC-60B exhibited two prominent effects on actin polymerization (Fig. 7B). First, UNC-60B inhibited the early phase of the polymerization, but not as strong as UNC-60A, suggesting that binding of UNC-60B to G-actin inhibited the nucleation step. Rapid decrease in the absorbance was observed immediately after the addition of salt. The levels of decrease were dependent on the concentration of UNC-60B. Although the reason for this effect is currently unknown, some conformational change on UNC-60B or actin might be induced by salt.

Second, as reported for some other ADF/cofilin family members, UNC-60B accelerated the polymerization rate. Delayed acceleration of the polymerization was observed at high concentration of UNC-60B (2.0 mol/mol of actin). At 2 h (7,200 s), 2 molar excess of UNC-60B decreased the amount of polymerized actin, while lower amounts of UNC-60B appeared to increase the amount of polymerized actin. However, when these samples were examined by pelleting assay at 140,000 \( \times g \), the amounts of pelletable actin were not different up to 1 mol of
UNC-60B/mol of actin, while 2 molar excess of UNC-60B decreased pelletable actin (data not shown). The results suggest that F-actin, which was associated with UNC-60B, elongates rapidly although the high concentration of UNC-60B simultaneously binds to G-actin to inhibit polymerization.

**DISCUSSION**

The present study shows that the two ADF/cofilin proteins that are generated by alternative splicing of the unc-60 gene have differential functions in regulating actin dynamics. This is the first clear demonstration of functional diversity for the ADF/cofilin family in a single organism. So far, 3 and 2 ADF/cofilin proteins have been found in mammals and chickens, respectively, but their functional differences are not clear. Previously, ADF (or destrin) and cofilin had been characterized as functionally distinct proteins. ADF primarily depolymerizes F-actin and does not bind to F-actin (3, 21, 26, 27), whereas cofilin binds to both G- and F-actin and depolymerizes F-actin in a pH-sensitive manner (4, 16, 28). However, recent refined biochemical studies on the activities of human and chicken ADF have shown that ADF, like cofilin, binds to F-actin at pHs between 6.5 and 7.1 and shows increasing actin-depolymerizing activity at pHs between 7.1 and 8.0 (24, 25).

Although the activities of UNC-60A and B are different, both of them are functionally related to members of the ADF/cofilin family that have been characterized previously. The actin-depolymerizing activity of UNC-60A is quite similar to that of Acanthamoeba actophorin (29), echinoderm depactin (20), and most of ADF/cofins at alkaline pH. In addition, the profile of the effect of UNC-60A on actin polymerization kinetics is similar to that of chicken ADF (3). These activities can be explained by its strong G-actin binding ability that results in depolymerization of F-actin and sequestering G-actin to inhibit polymerization. The ability of UNC-60B to bind to F-actin has been observed for vertebrate ADF and cofilin at neutral pH (4, 16, 24, 25). However, its lack of ability to depolymerize actin is a unique feature of UNC-60B. The effect of UNC-60B on the kinetics of actin polymerization is consistent with that observed for actophorin (29), chicken cofilin (4), and plant ADF.
Our results that inorganic phosphate inhibits the activities of both UNC-60A and B strongly suggest that UNC-60A and B preferentially bind to ADP-actin rather than ATP-actin. Preferential binding to ADP-actin appears to be a common property of ADF/cofilin family members (7, 22, 35) and is likely to contribute to an acceleration of the off rate at the pointed end of F-actin (7). Probably, this is the case for UNC-60A because it rapidly depolymerizes F-actin. However, because UNC-60B does not depolymerize F-actin, it implies that UNC-60B-bound F-ADP-actin is physiologically significant. Therefore, it is of interest to examine whether UNC-60B-bound F-actin behaves differently from F-actin alone. Previously, porcine cofilin has been shown to inhibit the binding of tropomyosin and myosin to F-actin (16). Thus, a future goal will be to investigate the ability of UNC-60B-bound F-actin to bind to some other actin-binding proteins.

The differences in the activities of the UNC-60 proteins are likely to result from their structural differences. The sequence identity between UNC-60A and UNC-60B is 36%, which is considerably lower than the group of mammalian ADF/cofilins (three members are 70% identical). However, both UNC-60A and UNC-60B are about 30% homologous to all three members of mammalian ADF/cofilins, and no outstanding similarity to a particular protein was detected. One obvious difference is that UNC-60A is 13 amino acids longer than UNC-60B. The alignment of the two sequences shows that an extra eight amino acids exist in UNC60A from Ile-50 to Asp-57 (Fig. 1, shown by underline). The equivalent region of yeast cofilin consists of the outer most strand of β-sheet and is exposed on the surface of the molecule (30), but the function of this region is unknown. In addition, this sequence contains four acidic residues, which are likely to affect the charge distribution on the molecular surface. Because charged amino acids on cofilin have been shown to be important in its actin-binding (31, 32), the extra sequence in UNC-60A may be responsible for its functional difference from UNC-60B.

The differential activities of UNC-60 proteins presented here strongly suggest that the two homologous proteins have physiologically distinct functions. The fine structure genetic map (13) and our preliminary genomic sequencing of viable unc-60 alleles has revealed that all the mutations are found within the coding region for unc-60B, implying that UNC-60B has a specific role in thin filament assembly in muscle cells. Our results on the effect of UNC-60B on the polymerization kinetics in vitro suggests that, during muscle development, when actin concentration is low initially, UNC-60B inhibits actin polymerization, but later, when actin concentration is high (1), UNC-60B accelerates actin polymerization and thus the formation of thin filaments. This function of UNC-60B is directly relevant to that of ADF/cofilin in vertebrate muscle. ADF/cofilin is involved in the regulation of actin assembly in chicken embryonic muscles (3, 4). In addition, a muscle-type cofilin isoform is expressed in mammalian skeletal and cardiac muscles (10) although its function in muscle cells is not yet clear. UNC-60A may be widely involved in the many processes which require actin dynamics. ADF/cofilin has been shown to be essential for cytokinesis (33, 34) and endocytosis (9), which are universal events in a broad range of cells. Accordingly, C. elegans should have an ADF/cofilin which is expressed in a variety of cells in addition to a muscle-specific isoform. Currently, we are raising antibodies against UNC-60A and UNC-60B and plan to determine tissue distribution of both proteins by immunofluorescence microscopy.

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