A- and B-utrophin Have Different Expression Patterns and Are Differentially Up-regulated in mdx Muscle*

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Duchenne muscular dystrophy (DMD) is a fatal childhood disease caused by mutations that abolish the expression of dystrophin in muscle. Utrophin is a paralogue of dystrophin and can functionally replace it in skeletal muscle. A method to induce utrophin up-regulation in muscle should therefore be therapeutically useful in DMD. We have previously shown that there are two full-length utrophin mRNA species: A and B. Here we describe the generation and characterization of antibodies specific to A- and B-utrophin. We show that both mRNA isoforms are translated into full-length proteins, which have very different expression patterns. B-utrophin is expressed in vascular endothelial cells; A-utrophin is expressed at the neuromuscular junction, choroid plexus, pia mater, and renal glomerulus. We have analyzed the expression of A- and B-utrophin protein and RNA in dystrophin-deficient tissues. We conclude that (i) the previously described expression patterns of utrophin represent a composite of A- and B-utrophin, (ii) A- but not B-utrophin is up-regulated in dystrophin-deficient striated muscle, and (iii) this up-regulation occurs post-transcriptionally with an additional transcriptional component in skeletal muscle. These results have important implications for understanding the biology of utrophin and are crucial for future studies aiming to effect its therapeutic up-regulation in DMD patients.

Utrophin, a large (376-kDa) cytoskeletal protein, is an autosomal paralogue of dystrophin and has similar binding partners and structure (1–5). Interest in the biology of utrophin has been enhanced by a potential role for the protein in novel cellular responses (10–12). An alternative approach, avoiding these difficulties, may be to develop a pharmacological means to up-regulate utrophin in muscle in an attempt to take advantage of the functional redundancy between dystrophin and utrophin. Proof of concept for this notion derives from experiments using mdx mice (a dystrophin-deficient model for DMD) harboring a transgene that increases utrophin levels in muscle. These mice are spared the pathological consequences of dystrophin deficiency (13, 14). The mechanisms controlling the expression of utrophin therefore assume considerable importance.

Analysis of the regulation of utrophin requires knowledge of the expression pattern of the protein. Utrophin is expressed ubiquitously but in a complex pattern within different adult tissues. In skeletal muscle, utrophin is found at the peripheral nerves, the vasculature, and the neuromuscular junction (NMJ) but not the extrajunctional sarcolemma; in the brain it is present at the pia mater, the choroid plexus, and blood vessels; and in the kidney it is expressed at the podocytes of the glomerulus and at some tubules and blood vessels (15–20). In DMD and mdx striated muscle, the distribution of utrophin changes so that it is expressed throughout the sarcolemma (16, 21–23).

We have recently shown that the utrophin mRNA population contains two full-length species (named A- and B-utrophin); these are both quantitatively significant, have different initial exons, and are transcribed from different promoters (24). The predicted protein sequences arising from these transcripts differ at their N termini (with unique sections of 31 and 26 amino acids, respectively). This raises the possibility that the expression pattern of utrophin represents a composite of A- and B-utrophin. Here we report the generation of antibodies specific to the unique epitopes predicted by the A- and B-utrophin sequences. We show that both transcripts are translated and that the expression patterns of A- and B-utrophin are different and change differentially in dystrophin-deficient muscle.

EXPERIMENTAL PROCEDURES

Antibodies—Antibodies UtroA and UtroB were made by immunizing New Zealand White rabbits with synthetic peptides pepA (MAKYGDLEARPDGQNEFSDDHKSRC) and pepB (MSSLTAATTFRWKKWRLDLPQVPLQAC) coupled to keyhole lymphocyte hemocyanin (Sigma). The resultant antiserum was affinity purified using the immunizing peptide immobilized on a SulfoLink column (Pierce) according to the manufacturer’s protocol. Antibody URD40 was raised against the distal rod domain of utrophin (25). MEC13.3 (BD Pharrmingen) is a rat monoclonal antibody that binds platelet endothelial cell adhesion molecule 1 (CD31) (26). mAb 1A4 (ICN) is a mouse monoclonal antibody that binds smooth muscle α-actin (27).

Animals—C57BL/10, mdx (28), and utrn−/− (29) mice aged 2–3 months were used.

Immunohistochemistry—Dissected tissues, embedded in OCT (Sakura), were frozen on liquid nitrogen-cooled isopentane. 10-μm cryosections were cut and kept at −70 °C. Sections were air dried and fixed.
in 2% paraformaldehyde in phosphate-buffered saline at 4 °C, washed three times for 5 min, and then processed as described in Ref. 19 using primary antibody at dilutions 1:10 (UtroA and UtroB) or 1:100 (URD40) and secondary antibody Alexa-586-conjugated goat anti-rabbit IgG F(ab')2 fragment (Molecular Probes) at dilution 1:200. To visualize the NMJ Alexa-488-conjugated α-bungarotoxin (Molecular Probes) was added to the diluted secondary antibody at dilution 1:100. As controls, sections were processed without primary antibody, with primary antibody preincubated with 100 μM antigenic peptide, and with utrn−/− sections. These controls were negative. Photomicrographs were acquired with a Leica DM LD fluorescent microscope and camera using fixed exposure times. Slides were digitally scanned with a QuickScan 35 (Minolta) as 1293×861-pixel TIF files.

**Dual Labeled Confocal Microscopy**—Dual labeling with MEC13.3 was carried out as above with the addition of the rat monoclonal (diluted 1:200) to the diluted primary antibody and of Alexa-488-conjugated goat anti-rabbit IgG (diluted 1:200) to the diluted secondary antibody. Dual labeling with mAb 1A4 was carried out by first applying the mouse monoclonal antibody and fluorescein-conjugated secondary antibody using the M.O.M. immunodetection kit (Vector) following the manufacturer’s protocol and then labeling with UtroA or UtroB as described above. Laser scanning confocal microscopy was performed with a Leica DMRE TCS SP microscope. To guard against bleed-through between channels, controls were performed omitting each primary antibody one at a time.

**Immunoblotting**—Dot blots were made by spotting 2 μg of peptides in 2% paraformaldehyde in phosphate-buffered saline at 4 °C, washed three times for 5 min, and then processed as described in Ref. 19 using primary antibody at dilutions 1:10 (UtroA and UtroB) or 1:100 (URD40) and secondary antibody Alexa-586-conjugated goat anti-rabbit IgG F(ab')2 fragment (Molecular Probes) at dilution 1:200. To visualize the NMJ Alexa-488-conjugated α-bungarotoxin (Molecular Probes) was added to the diluted secondary antibody at dilution 1:100. As controls, sections were processed without primary antibody, with primary antibody preincubated with 100 μM antigenic peptide, and with utrn−/− sections. These controls were negative. Photomicrographs were acquired with a Leica DM LD fluorescent microscope and camera using fixed exposure times. Slides were digitally scanned with a QuickScan 35 (Minolta) as 1293×861-pixel TIF files.

**A- and B-utrophin in Normal and mdx Tissues**

**Fig. 1.** Characterization of antibodies UtroA and UtroB. a, Western blot performed with 80 μg of protein (heart) per lane and probed in a Deca-Probe incubation manifold (Amersham Biosciences) with UtroA (A), UtroB (B), and URD40 (U). The lower panel shows a gel run in parallel stained with Coomassie Blue. b, two peptide dot blots, each made with peptides pepA and pepB, probed with UtroA (panel A) and UtroB (panel B). c, Western blot performed with 24 μg of protein from wild-type (wt) or utrn−/− heart and probed with UtroA. The lower panel shows the membrane stained with SYPRO Ruby blot stain to demonstrate equal loading. d, Western blot performed with 24 μg of protein from wild-type or utrn−/− heart and probed with UtroB. The lower panel shows the membrane stained with SYPRO Ruby blot stain to demonstrate equal loading. e, Western blot performed with 20 μg of protein (tibialis anterior) and probed with UtroA or URD40 with and without preincubation with pepA. f, Western blot performed with 20 μg of protein (tibialis anterior) and probed with UtroB or URD40 with and without preincubation with pepB. Solid arrowhead, 220-kDa marker; gray arrowhead, 66-kDa marker; open arrowhead, 46-kDa marker.
pepA and pepB onto a nitrocellulose membrane. Protein was extracted from cells and tissues, and Western blotting was performed as in Ref. 30 with primary antibodies UtroA (dilution 1:30), UtroB (1:100), and URD40 (1:100). For quantitative studies, equal loading and transfer of samples was ensured by staining nitrocellulose membranes after transfer with SYPRO Ruby blot stain (Molecular Probes) using the manufacturer’s protocol and scanning using a Typhoon imager (Amersham Biosciences). The total protein transferred to the membrane in each sample was quantified using ImageQuant and varied by less than 20% between lanes.

Ribonuclease Protection—The ribonuclease protection assay enabling specific quantitative determination of A- and B-utrophin mRNA levels was described previously (24). Total RNA was extracted from mouse tissues using Ultraspec (Biotecx) following the manufacturer’s protocol and scanning using a Typhoon imager (Amersham Biosciences). The total protein transferred to the membrane in each sample was quantified using ImageQuant and varied by less than 20% between lanes.

RESULTS

Both A- and B-utrophin Are Translated—To distinguish A- and B-utrophin, we raised antibodies (UtroA and UtroB) against peptides with the sequences of the predicted unique N termini. Western blot analysis showed that both antibodies recognized single bands with the same molecular weight as utrophin (Fig. 1a); these bands were absent from blots (Fig. 1, c and d) made with the tissues of utrn−/− mice (which lack both utrophin isoforms). Specificity of UtroA and UtroB for their cognate peptides was confirmed by dot blotting (Fig. 1, e and f). Taken together these data demonstrate that antibodies UtroA and UtroB are specific for A- and B-utrophin and that both A- and B-utrophin mRNAs are translated into full-length protein isoforms.

A- and B-utrophin Have Complementary Distributions in Skeletal Muscle—Immunohistochemistry using the isoform-specific antibodies UtroA and UtroB demonstrated that the...
A- and B-utrophin in Normal and mdx Tissues

FIG. 5. Distribution of A- and B-utrophin within blood vessels. Dual labeled confocal images. a, endomysial capillaries in a section of tibialis anterior stained using UtroA (I) (red) and the endothelial marker MEC13.3 (II) (anti-CD31, green). III shows images I and II merged. b, two serial sections of an arteriole in tibialis anterior stained using UtroB (red, I and II) and either the endothelial marker MEC13.3 (I) (anti-CD31, green) or the smooth muscle marker mAb 1A4 (II) (anti-vascular smooth muscle α-actin, green). c, five serial sections of a coronary artery stained using UtroA only (red) (I), UtroA (red) and mAb 1A4 (green) (II), mAb 1A4 only (green) (III), UtroB (red) and mAb 1A4 (green) (IV), or UtroB only (red) (V). Bar, 50 μm.

distributions of A- and B-utrophin in skeletal muscle are different (Figs. 2 and 3). A-utrophin localizes to the NMJs and peripheral nerves; weak staining of arterioles was also observed. In contrast, B-utrophin was observed only in endomysial capillaries and other blood vessels.

A- and B-utrophin in Other Tissues—To test whether the vascular localization of B-utrophin was a general phenomenon, we examined other tissues immunohistochemically using antibodies UtroA, UtroB, and URD40 (Fig. 4). In the myocardium, kidney, and brain, B-utrophin was confined to blood vessels. As in skeletal muscle, the distribution of A-utrophin was largely complementary to B-utrophin; antibody UtroA bound to the glomerular tufts and tubules in the renal cortex and the choroid plexus and pia mater in the brain; weak staining was also seen in larger blood vessels in the heart and some small vessels in the brain. We have not observed any structures expressing utrophin (as revealed by binding of the nonspecific utrophin antibody URD40) that do not bind either UtroA or UtroB.

B-utrophin Localizes to the Vascular Endothelium—To define more precisely which elements of blood vessels express B-utrophin, we examined arterioles using dual labeled confocal immunofluorescence with markers of vascular endothelium (CD31) and smooth muscle (α-actin) (Fig. 5b). B-utrophin expression was distinct from, and intimal to, the smooth muscle layer and co-localized with CD31 at the endothelium. B-utrophin also co-localized with the endothelial marker in capillaries (Fig. 5c).

As vascular UtroA staining in the heart and skeletal muscles was seen only in larger blood vessels, we hypothesized that this signal derived from A-utrophin expression in vascular smooth muscle. Confocal imaging showed that A- but not B-utrophin co-localized with the smooth muscle marker in arteries (Fig. 5c).

A-, but Not B-, Utrophin Is Up-regulated in Dystrophin-deficient Muscle—We next asked whether the up-regulation of utrophin that occurs in dystrophin-deficient muscle affects both A- and B-utrophin. Previous reports have suggested that this phenomenon is restricted to muscle cells; we therefore hypothesized that A-, but not B-, utrophin would be up-regulated in mdx muscle. Immunohistochemistry confirmed that the wild-type expression pattern of A-utrophin (Fig. 6a) was altered in mdx skeletal muscle in that the protein was widely distributed along the sarcolemma (Fig. 6c), whereas the distribution of B-utrophin was unchanged with respect to the control (Fig. 6, b and d). In the heart, very little staining for A-utrophin was observed in controls, but this was greatly enhanced in mdx heart where A-utrophin was localized to the sarcolemma of cardiomyocytes (Fig. 6, e and g). In contrast, the endothelial distribution of B-utrophin in the heart was unchanged in mdx heart compared with controls (Fig. 6, f and h).

A-utrophin Up-regulation in Skeletal and Cardiac Muscle Is Not Accompanied by Commensurate Increases in A-utrophin mRNA—To assess whether the increases in A-utrophin protein occurring in skeletal and cardiac muscle were accompanied by increases in A-utrophin mRNA, we performed quantitative ribonuclease protection analysis on total RNA isolated from control and mdx skeletal and cardiac muscles (Fig. 7). A-utrophin mRNA levels were 50% higher in mdx skeletal muscle than in controls, whereas levels of B-utrophin mRNA were unchanged. The levels of both A- and B-utrophin mRNA were unchanged between control and mdx heart.

DISCUSSION

We generated utrophin isoform-specific antibodies, a crucial resource that allowed us to ask a number of important questions about the expression patterns of A- and B-utrophin in healthy and dystrophic tissues. The results presented here are important because of their implications both for the biological roles of utrophin and for further studies of utrophin regulation.

Our data show that the previously described expression pattern of utrophin represents a composite of the very different expression profiles of A- and B-utrophin. B-utrophin expression is restricted to the vascular endothelium. In contrast, A-utrophin is expressed in a variety of structures, including the NMJ, peripheral nerves, and vascular smooth muscle. A-utrophin was not seen in association with capillaries except in the brain and that at low levels; this could derive from either the endothelium or the associated astrocyte foot processes, which have both been found to contain utrophin in previous studies (17, 18). The localization of A-utrophin at the NMJ is in accordance with previous work suggesting that transcription from promotor A is enhanced at subjunctional nuclei underlying the postsynaptic membrane, consequent to activation by nerve-derived signals (32). We previously noted that promoter B does not contain the relevant cis-acting sequences to mediate synaptically enhanced transcription and hypothesized that muscle B-utrophin might be localized to either (i) the extrajunctional region of the sarcolemma or (ii) other cell types in muscle (24).

The present findings enable us to reject the first hypothesis and accept the second; B-utrophin is the vascular endothelial isoform of utrophin.

The expression of utrophin in vascular endothelium has previously been recognized (33); the finding that the B-isoform is
specific for this structure suggests that it may have an important endothelial function. Mice lacking utrophin have abnormal NMJs; other gross pathology has not been identified, but it is possible that a subtle phenotype results from defects of endothelial function (29, 34). Key to the functions of utrophin and dystrophin are their interactions with actin and with the dystrophin/utrophin associated protein complex (35). Members of this group present in at least some endothelial cells include dystroglycan, α-dystrobrevin, and the syntrophins, and these molecules are therefore candidate members for an endothelial B-utrophin associated protein complex (19, 25, 36–39).

The interaction of utrophin with actin is mediated via the N terminus, which contains the region that differs in A- and B-utrophin. Indeed the A-utrophin unique region forms part of the actin binding site (4, 40, 41). It may be therefore that the interactions between actin and A- and B-utrophin are functionally different; this may have important consequences for the therapeutic up-regulation of utrophin in DMD skeletal muscle as the functional redundancy between dystrophin and utrophin was shown using an A-utrophin transgene. It will be necessary to demonstrate that B-utrophin can also replace dystrophin in skeletal muscle before any strategy seeking to up-regulate utrophin in muscle by stimulation of promoter B can be contemplated.

The development of resources allowing the characterization and measurement of A- and B-utrophin at both the mRNA and protein levels enables us to clarify an important issue regarding the expression of utrophin in dystrophin-deficient muscle. Previous reports have not distinguished between the two full-length isoforms of utrophin and have suggested that up-regulation of utrophin in dystrophic muscle is unaccompanied by changes in steady-state mRNA levels (42). Here we unequivocally demonstrate that dystrophin deficiency in the heart and skeletal muscles is associated with up-regulation of B-utrophin and redistri-

![Fig. 6. Changing distribution of A-utrophin in mdx striated muscle.](image)

![Fig. 7. Quantification of utrophin levels in wild-type (wt) and mdx striated muscle using Western blots, SYPRO Ruby blot stained membranes (to demonstrate equal loading), and densitometry.](image)
tion; we have recently shown that A-utrophin is transcriptionally induced during early myogenesis through activation of promoter A by myogenic factors (31). However, this is unlikely to account for the magnitude of the change in protein expression seen in skeletal muscle, which is disproportionate to the small change in RNA level. Furthermore, regeneration occurs at very low levels in the heart and cannot therefore account for the increase in A-utrophin expression seen in the mdx mouse myocardium, which occurs in the absence of an increase in A-utrophin mRNA levels. These data strongly suggest involvement of additional post-transcriptional mechanisms in the regulation of A-utrophin expression in the heart and in muscle.

We conclude that it is essential to distinguish between A- and B-utrophin in future studies examining the regulation and putative therapeutic applications of these proteins. Characterization of the factors governing the distinct expression patterns of the two full-length isoforms may permit definition of mechanisms that could be manipulated to effect up-regulation of utrophin and thereby help achieve a much-needed therapeutic option for patients with DMD.

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