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Heteromeric TRPC3 with TRPC1 formed via its ankyrin repeats regulates the resting cytosolic Ca^{2+} levels in skeletal muscle

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ABSTRACT

The main tasks of skeletal muscle are muscle contraction and relaxation, which are mediated by changes in cytosolic Ca^{2+} levels. Canonical-type transient receptor potential 3 (TRPC3) contains an ankyrin repeat (AR) region at the N-terminus (38–188 amino acids) and forms extracellular Ca^{2+}-entry channels by homo or heteromerization with other TRP subtypes in various cells including skeletal myotubes. However, previous research has not determined which region(s) of TRPC3 is responsible for the heteromerization, whether the AR region participates in the heteromerizations, or what is the role of heteromeric TRPC3s in skeletal muscle. In the present study, the heteromerization of TRPC3 with TRPC1 was first examined by GST pull-down assays of TRPC3 portions with TRPC1. The portion containing the AR region of TRPC3 was bound to the TRPC1, but the binding was inhibited by the very end sub-region of the TRPC3 (1–37 amino acids). In-silico studies have suggested that the very end sub-region possibly induces a structural change in the AR region. Second, the very end sub-region of TRPC3 was expressed in mouse primary skeletal myotubes, resulting in a dominant-negative inhibition of heteromeric TRPC3/1 formation. In addition, the skeletal myotubes expressing the very end sub-region showed a decrease in resting cytosolic Ca^{2+} levels. These results suggest that the AR region of TRPC3 could mediate the heteromeric TRPC3/1 formation, and the heteromeric TRPC3/1 could participate in regulating the resting cytosolic Ca^{2+} levels in skeletal muscle.

1. Introduction

The transient receptor potential (TRP) super-family consists of a diverse group of Ca^{2+}-permeable non-selective cation channels on the plasma membrane, and plays important roles in extracellular Ca^{2+}-entry into various cells [1]. Tetrameric complexes of TRPs function as the channels, and the formation of tetrameric complexes is accomplished by homo or heteromerization of TRPs of either the same or different subfamilies [2]. Canonical-type TRP (TRPC) is a sub-family of the TRP super-family, and seven TRPC3 subtypes have been known [1]. Each TRPC subtype has six transmembrane domains, the N-terminus containing an ankyrin repeat (AR) region, and the C-terminus containing a TRP domain and a proline-rich motif [1,3].

Ca^{2+} stored in the sarcoplasmic reticulum is the major Ca^{2+} source for the excitation–contraction coupling (ECC) of skeletal muscle [4,5]. By allowing extracellular Ca^{2+}-entry, TRPC3 in skeletal myotubes maintains a sustained high Ca^{2+} level in the cytosol to allow a full gain during ECC [4,6]. TRPC3 binds to ECC-mediating proteins in skeletal myotubes, such as ryanodine receptor 1 (RyR1) [7,8], and plays an important role in the proliferation and differentiation of skeletal myoblasts to myotubes [6,8,9]. TRPC3 is also closely related to skeletal muscle disease. Patients with myasthenia gravis show fluctuating muscle weakness and fatigue due to the circulation of auto-antibodies against TRPC3 [10]. TRPC3-overexpressing transgenic mice show a phenotype of Duchenne muscular dystrophy (DMD) involving muscle weakness, wasting, and premature death [11]. A mouse model of atrophy shows a decrease in TRPC3 expression [12].

Ankyrin repeat (AR) is an interface for protein–protein interactions and is involved in a wide variety of physiological and pathophysiological processes via the mediation of local and short-range protein–protein interactions [13]. AR consists of 30–34 amino acids, and each repeat contains an anti-parallel helix-turn-helix

Abbreviations: TRP, transient receptor potential; TRPC, canonical-type TRP; AR, ankyrin repeats; ECC, excitation–contraction coupling; DMD, Duchenne muscular dystrophy; 3D, three-dimensional.

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structure followed by a flexible loop leading to the turn of the next repeat, all of which are stacked together to form a single domain [13,14]. An AR region consisting of four ARs exists in the N-terminus of TRPC3, and is required for the correct targeting of TRPC3 to the plasma membrane [15]. On the other hand, TRPC3 is heteromerized with other TRPC subtypes to form functional channels [2]. Among them, the heteromerization of TRPC3 with TRPC1 is found in both heterologous expressions and bona-fide systems including skeletal myotubes [2,16–18]. Through yeast two-hybrid analysis, an interaction between the N-termini from TRPC3 and TRPC1 has been reported [16]. However, there have been no studies to determine more specific region(s) in the N-terminus of TRPC3 that could be responsible for the heteromerization with TRPC1. In addition, no study has addressed whether the AR region participates in the heteromerization or what is the role of heteromeric TRPC3s in skeletal muscle, although heteromeric TRPC3s are known to exist in skeletal muscle [18], and the presence of the AR region is a characteristic of the TRPC family [1,3]. Therefore, in the present study, we examined the role of the AR region of TRPC3 in the heteromeric TRPC3/1 formation and the role of the heteromeric TRPC3/1 in skeletal myotubes.

2. Materials and methods

2.1. Ethics statement

All surgical interventions and all pre- and post-surgical animal care were provided in accordance with the Laboratory Animals Welfare Act, the Guide for the Care and Use of Laboratory Animals, and the Guidelines and Policies for Rodent Survival Surgery provided by the Institutional Animal Care and Use Committee of the College of Medicine at The Catholic University of Korea.

2.2. cDNA construction and protein expression of GST-fused TRPC3 portions

Using full-length human TRPC3 cDNA (GenBank Accession No. NM_003305.2) as a template, the N-terminal portions of TRPC3 were synthesized by PCR using the primers presented in Supplementary data 1. The PCR products were inserted into pGEX-4T-1 vector at the EcoRI and SalI sites (GST-fused NF1–NF4, or NF5) or into pEGFP-C1 mammalian expression vector at the XhoI and SalI sites (GFP-NF4). The sequences of all constructs were confirmed by sequencing both strands using an ABI Prism 3700 DNA Sequencer (Applied Biosystems, Foster City, CA). Each GST-fused protein was expressed in *Escherichia coli* (DH5α), as previously described [19].

2.3. Preparation of the triad sample and the GST pull-down assay of TRPC3 portions with TRPC1

The triad vesicles containing TRPC1 were prepared from rabbit fast-twitch skeletal muscle and were solubilized, as previously described [7,20]. GST pull-down assays were performed as previously described [19]. Briefly, affinity beads were prepared by immobilizing each GST-fused TRPC3 portion on GST beads (Amersham Biosciences, Pittsburgh, PA). The affinity beads were then incubated with 150 μg of the solubilized triad sample for 8 h at 4 °C. The proteins that were pulled down were separated on SDS–PAGE gels and subjected to immunoblot assay.

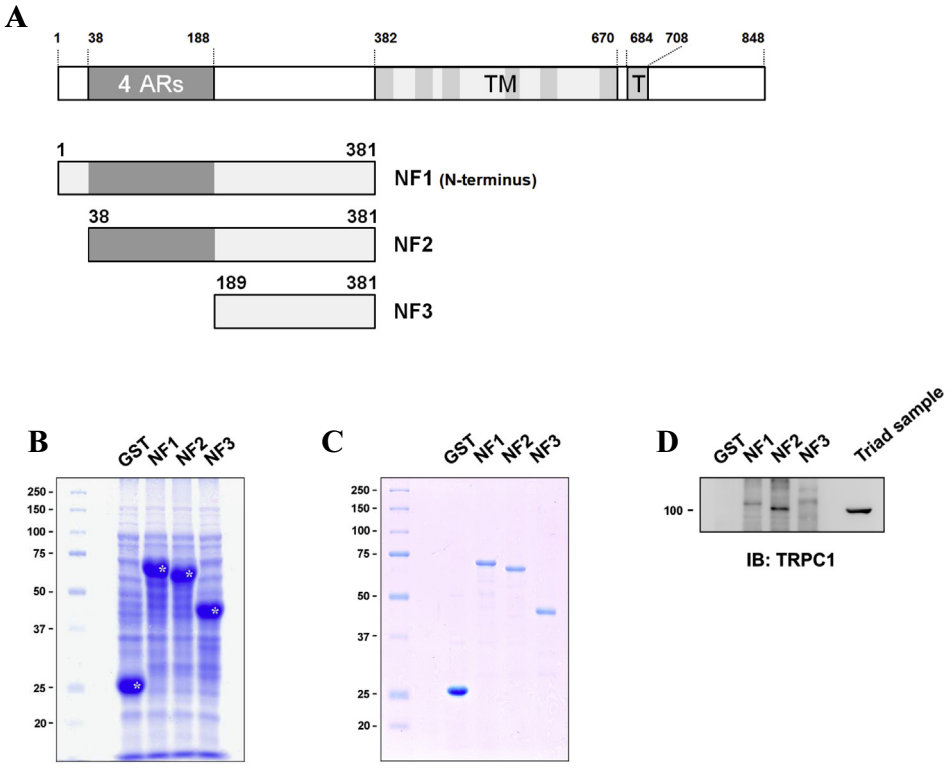

Fig. 1. Schematic primary sequences of TRPC3 and TRPC3 portions, and GST pull-down assays of the TRPC3 portions with TRPC1. (A) Schematic diagrams of full-length TRPC3 and N-terminal TRPC3 portions. Numbers indicate the sequence of amino acids. AR, ankyrin repeat; TM, transmembrane domain; T, TRP domain. (B) Bacterial lysate expressing each GST-fused TRPC3 portion was separated on SDS–PAGE gel, and the gel was stained with Coomassie Blue. GST-fused TRPC3 portions are indicated by asterisks. (C) Immobilized GST-fused TRPC3 portions on GST beads were separated on SDS–PAGE gel, and the gel was stained with Coomassie Blue. (D) Proteins obtained from the GST pull-down assays of GST-fused TRPC3 portions with TRPC1 were separated on SDS–PAGE gel, and the gel was subjected to immunoblot assay with anti-TRPC1 antibody. GST was used as a negative control. Three independent experiments were conducted and a representative result is presented. Only NF2 would bind to TRPC1.

2.4. Cell culture and cDNA transfection

Mouse primary skeletal myoblasts were derived from mouse skeletal muscle, then were allowed to proliferate and differentiate to myotubes, as previously described [21–23]. For the transfection of NF4 cDNA, after 3 days of culture in the differentiation medium, the immature myotubes were transfected with NF4 cDNA (with a mixture of 30 μl of FuGENE6 transfection reagent (Promega, Madison, WI) and 20 μg of cDNA per 10-cm plate or the same ratio of the components in the wells of 96-well plates), as previously described [21,23]. On differentiation day 5 (at 38 h post-transfection), the fully differentiated myotubes were subjected to further experiments. The successful expression of NF4 was confirmed using immunocytochemistry (approximately 50%).

2.5. Measurement of resting cytosolic Ca^{2+} levels

Mouse primary skeletal myotubes in 96-well plates were loaded with 5 μM of fura-2 in an imaging solution (125 mM NaCl, 5 mM KCl, 2 mM KH_2PO_4, 2 mM $CaCl_2$, 25 mM HEPES, 6 mM glucose, 1.2 mM $MgSO_4$, and 0.05% BSA (fraction V) at pH 7.4.) at 37 °C for 45 min. The loaded myotubes were subjected to single myotube Ca^{2+} imaging experiments using an inverted microscope equipped with a Nikon 40× oil-immersion objective (ECLIPSE Ti, Nikon Instruments), a high-speed monochromator with a 75W xenon lamp (FSM150Xe, Bentham Instruments), and a 12-bit charge-coupled device camera (DVC-340M-OO-CL, Digital Video Camera Company), as previously described [21,24]. The fura-2 in the myotubes was excited at 340 and 380 nm, and the fluorescence emission was measured at 510 nm. The data were displayed and analyzed using image acquisition and analysis software (High-Speed InCyt Im2, v5.29, Intracellular Imaging). The Ca^{2+} concentrations were calculated using 225 nM as the Ca^{2+}-fura-2 dissociation constant. All reagents for Ca^{2+} imaging experiments were obtained from Sigma–Aldrich (St. Louis, MO).

2.6. Co-immunoprecipitation, immunoblot assay, and immunocytochemistry

For co-immunoprecipitation, the mouse primary skeletal myotubes transfected with NF4 cDNA on 10-cm plates were solubilized, and the 800 μg total protein of the solubilized lysate and anti-TRPC3 antibody (10 μg/ml, Alomone Labs, Jerusalem, Israel) were used, as previously described [7]. For immunoblot assay, anti-TRPC3 (1:1000), anti-TRPC1 (1:1000, Santa Cruz Biotechnology, Dallas, TX), anti-Orai1 (1:1000, Abcam, Cambridge, MA), or anti-STIM1 (1:1000, Cell Signaling Technology, Danvers, MA) was used. For the SDS–PAGE, 10% gel was used. For immunocytochemistry, anti-GFP antibody (1:500, Invitrogen, Grand Island, NY) and FITC-conjugated secondary antibody (1:500, Sigma–Aldrich) were used. Other procedures were performed as previously described [21].

2.7. In-silico approaches and statistical analysis

Comparisons in the amino acid sequences of human TRPC3 and TRPC1 (GenBank Accession No. BC113953.1) were conducted using BLAST (http://blast.ncbi.nlm.nih.gov/Blast.cgi). The three-dimensional (3D) structure was predicted using RaptorX [25] and was

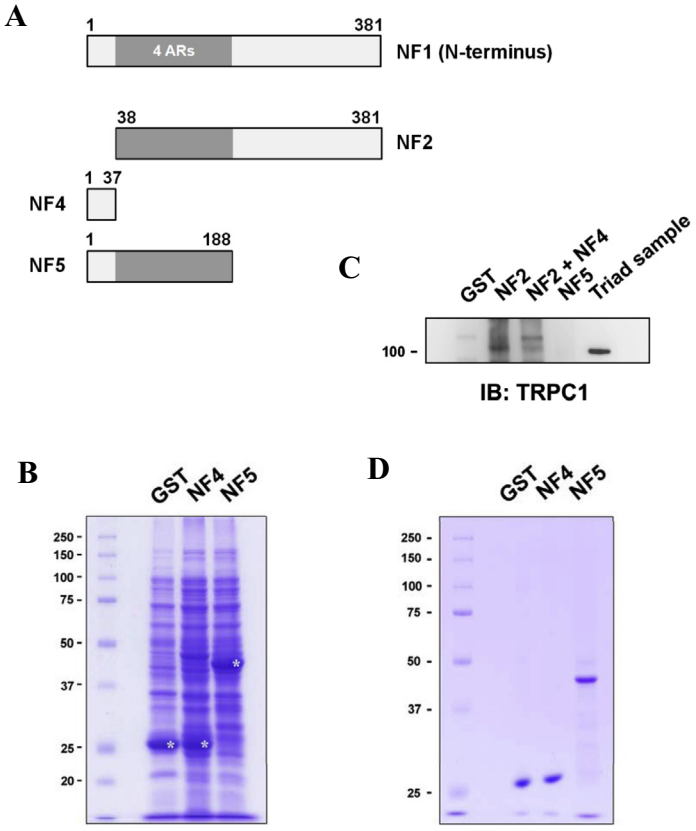

Fig. 2. GST pull-down assays of NF2 with TRPC1 in the presence of NF4, or NF5 with TRPC1. (A) Schematic diagrams of NF4 and NF5. Numbers indicate the sequence of amino acids. (B) Bacterial lysate expressing GST-fused NF4 or NF5 was separated on SDS–PAGE gel, and the gel was stained with Coomassie Blue. GST-fused NF4 or NF5 are indicated by asterisks. (C) Proteins obtained from the GST pull-down assays of NF4 with TRPC1 in the absence (NF2) or presence of NF4 (NF2 + NF4), or NF5 with TRPC1 (NF5) were separated on SDS–PAGE gel, and the gel was subjected to immunoblot assay with anti-TRPC1 antibody. GST was used as a negative control. The interaction between NF2 and TRPC1 was inhibited by the presence of NF4. NF5 did not bind to TRPC1. Three independent experiments were conducted and a representative result is presented. (D) Immobilized GST-fused NF4 or NF5 on GST beads was separated on SDS–PAGE gel, and the gel was stained with Coomassie Blue.

presented using a Jmol program (http://www.jmol.org). The results are presented as the means ± S.E. The number of experiments is presented in the figure legends. The significant differences were analyzed using a paired *t*-test (GraphPad InStat, v2.04, GraphPad Software, La Jolla, CA). The differences were considered to be significant at $p < 0.05$. The graphs were prepared using Origin v7 software (OriginLab, Northampton, MA).

3. Results and discussion

3.1. The AR region of TRPC3 binds to TRPC1

To investigate the role of the AR region in TRPC3 (38–188 amino acids) in the heteromeric TRPC3/1 formation, cDNAs for the three GST-fused N-terminal portions of TRPC3 were constructed (Fig. 1A): NF1 (full-length N-terminus), NF2 (shorter N-terminus starting from the AR region, i.e., with ARs), and NF3 (shorter N-terminus starting after the AR region, i.e., without ARs). Each GST-fused TRPC3 portion was expressed in *E. coli*, and the bacterial cell lysate was separated on SDS–PAGE gel and stained with Coomassie Blue. Each GST-fused TRPC3 portion was successfully expressed (Fig. 1B). To examine the binding of each GST-fused TRPC3 portion to TRPC1, GST pull-down assays were conducted using the immobilized TRPC3 portions on GST beads (Fig. 1C) and the solubilized triad sample from rabbit skeletal muscle. The triad samples were enriched with proteins that exist on the plasma and sarcoplasmic reticulum membrane, including TRPC1 [4,7]. The proteins that were pulled down were subjected to immunoblot assay with anti-TRPC1 antibody (Fig. 1D). A triad sample containing TRPC1 was loaded in order to indicate the exact position of the TRPC1 on the gel. The GST itself did not bind to the TRPC1. The NF2 possessing an AR region would bind to TRPC1, but the shorter one, NF3 lacking an AR region, did not bind to TRPC1, suggesting that the AR region of TRPC3 could be involved in the heteromeric TRPC3/1 formation. This is the first indication that the role of the AR region has more to do with the formation of heteromeric TRPC3 rather than with the function of TRPC3.

However, it is noteworthy that NF1, the full-length N-terminus possessing the AR region, did not bind to TRPC1. This result suggested the possibility that the binding of the AR region to TRPC1 was inhibited by the region(s) that exists only in NF1, which indicated that the N-terminal very end sub-region of TRPC3 was the candidate region that caused the inhibition.

3.2. The very end sub-region of TRPC3 at the N-terminus inhibits the binding of the AR region to TRPC1

The region that is a candidate for the inhibition (the very end sub-region of TRPC3 at the N-terminus, 1–37 amino acids) was constructed (Fig. 2A, NF4). GST-fused NF4 was expressed in *E. coli*, and the bacterial cell lysate was separated on SDS–PAGE gel and stained with Coomassie Blue. NF4 was successfully expressed (Fig. 2B). In the absence or presence of NF4, GST pull-down assays of NF2 with TRPC1 were conducted, as described in Fig. 1. As shown in Fig. 1D, NF2 itself (in the absence of NF4) was successfully bound to TRPC1, however NF2 in the presence of NF4 almost did not bind to TRPC1 (Fig. 2C). A triad sample containing TRPC1 was loaded in order to indicate the exact position of TRPC1 on the gel. In addition, NF5 (a continuous sequence of NF4 and the AR region, 1–188 amino acids of TRPC3) was constructed and was expressed in *E. coli* (Fig. 2B). GST pull-down assays of NF5 with TRPC1 were conducted (Fig. 2C). NF5 also did not bind to TRPC1. These results suggest that the very end sub-region of TRPC3 inhibited the binding of the AR region to TRPC1.

According to comparisons of the amino acid sequences, there was no significant similarity between the very end sub-region of TRPC3 and the remaining portion of TRPC3 or with any region of TRPC1. Therefore, the very end sub-region could be a unique region that inhibits the binding of the AR region to TRPC1.

3.3. The inhibitory action of the very end sub-region is possibly due to a structural change in the AR region

Based on the known 3D structures of ARs in other proteins, the 3D structure of the AR region from TRPC3 was predicted as

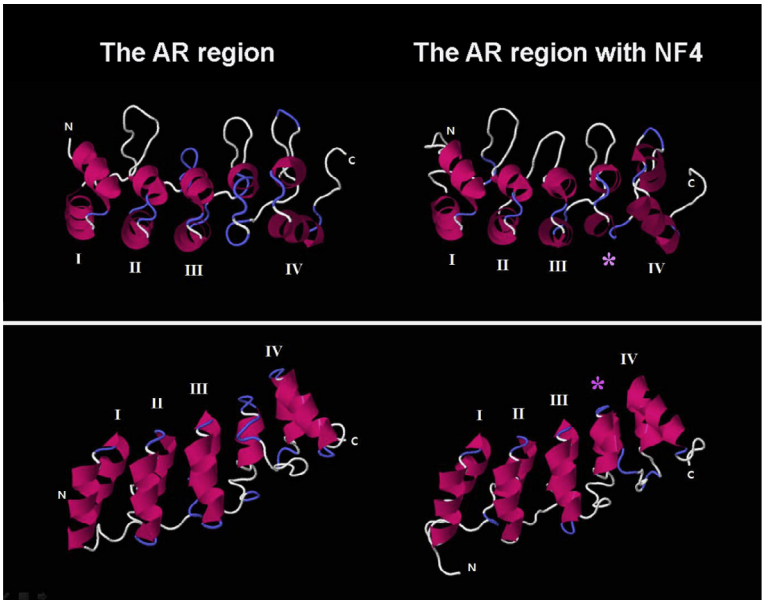

Fig. 3. The predicted 3D structure of the AR region from TRPC3. (A) The 3D structure of the AR region from TRPC3 in the absence (on the left-hand side, 38–188 amino acids) or presence of the very end sub-region (NF4) (on the right-hand side, 1–188 amino acids) was predicted using RaptorX [25]. Alpha-helix, beta-turn, or random coils are colored in red, blue, or white, respectively. N or C refers to the N- or C-terminus, respectively. Four ARs were numbered from I to IV. The newly appeared AR by the presence of the very end sub-region is indicated by an asterisk. The bottom images are the lateral view of the top images (−90° rotation of the top images along the horizontal axis). (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

described in the Section 2. Four ARs were predicted to exist in the AR region (I–IV, Fig. 3, on the left-hand side). It is noteworthy that the AR region in the presence of the very end sub-region (NF4) at the N-terminus (i.e., a continuous sequence from 1 to 188 amino acids of TRPC3) showed an additional AR between AR III and IV, forming five more evenly spaced ARs (indicated by an asterisk in Fig. 3, on the right-hand side). The very end sub-region was predicted to be unstructured either individually or within the continuous sequence. These in silico studies suggest the possibility that the structural change in the AR region by the very end sub-region could inhibit the binding of the AR region to TRPC1.

In addition, it is the presence, rather than the position, of the very end sub-region that could be the factor causing the structural change in the AR region, because an additional AR was also predicted by the presence of the very end sub-region at the opposite end of the AR region (at C-terminus, Supplementary data 2). This is in accordance with the facts showing no consensus amino acid sequence of AR that is universally recognized, but that AR is defined by its tertiary structure [14].

3.4. In skeletal myotubes, the very end sub-region dominant-negatively decreases the heteromeric TRPC3/1 formation

To examine the dominant-negative effect of the very end sub-region (NF4) on heteromeric TRPC3/1 formation in mammalian skeletal myotubes which express TRPC1 and TRPC3 by nature, NF4 cDNA was transfected to mouse primary skeletal myotubes as described in the Section 2. NF4 was successfully expressed in the myotubes (Fig. 4A). A co-immunoprecipitation assay of full-length TRPC3 with TRPC1 was conducted using the lysate from NF4-expressing skeletal myotubes with anti-TRPC3 antibody. Much less TRPC1 was co-immunoprecipitated with TRPC3 in NF4-expressing skeletal myotubes compared with the Vector control (Fig. 4B, more than 35% reduction), suggesting that the NF4 dominant-negatively inhibited the heteromeric TRPC3/1 formation in the skeletal myotubes. In order to rule out the possibility that the reduction was due to changes in the amount of TRPC1 and/or TRPC3 expression, those levels were examined by immunoblot analysis (Fig. 4C). Neither was affected by the NF4 expression.

3.5. Skeletal myotubes expressing the very end sub-region show a decrease in resting cytosolic Ca^{2+} levels

Based on the fact that TRPC3 is involved in regulating the resting cytosolic Ca^{2+} levels of myotubes from a mouse disease model of malignant hyperthermia [26], the resting cytosolic Ca^{2+} levels in the myotubes were examined. Resting cytosolic Ca^{2+} levels of NF4-expressing skeletal myotubes were significantly decreased compared with that of control myotubes (Fig. 4D). In order to rule out the possibility that the decrease was due to the other proteins,

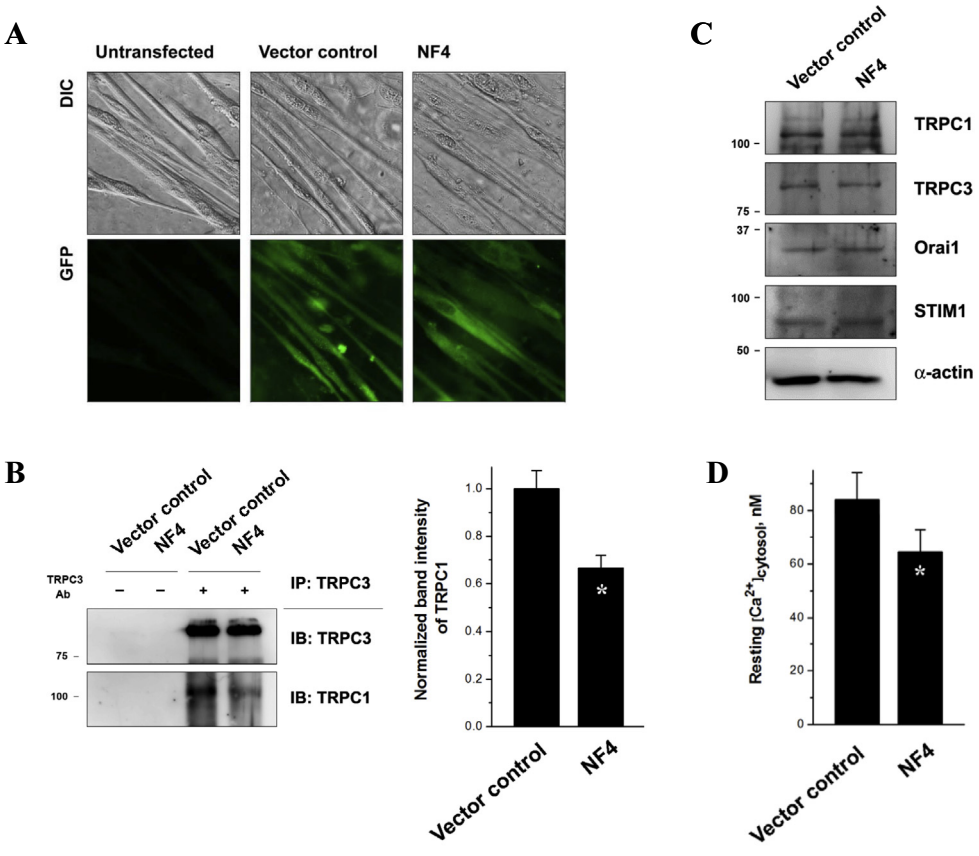

Fig. 4. Co-immunoprecipitation of full-length TRPC3 and TRPC1, and the measurement of resting cytosolic Ca^{2+} levels in mouse primary skeletal myotubes expressing the very end sub-region (NF4). (A) Mouse primary skeletal myotubes transfected with NF4 cDNA are presented. Vector control is the myotubes transfected with GFP vector. NF4 was successfully expressed in the myotubes. (B) Co-immunoprecipitation assay of full-length TRPC3 with TRPC1 was conducted using the lysate from the NF4-expressing myotubes with anti-TRPC3 antibody. Three independent experiments were conducted and a representative result is presented (on left-hand side). The band intensities of TRPC1 relative to TRPC3 in NF4 samples were normalized to those in Vector control samples (on right-hand side). Less TRPC1 was co-immunoprecipitated with TRPC3 in NF4-expressing myotubes compared with Vector control (0.67 ± 0.05 vs. 1.00 ± 0.08). (C) The lysate from NF4-expressing myotubes (50 μg of total protein) was subjected to immunoblot assay with various antibodies. α-Actin was a loading control. The expression levels of TRPC1, TRPC3, Orai1, or STIM1 were not affected by the NF4 expression. Three independent experiments were conducted and a representative result is presented. (D) Resting cytosolic Ca^{2+} levels in the NF4-expressing myotubes were measured. The resting cytosolic Ca^{2+} level was significantly decreased in the NF4-expressing myotubes compared with those from Vector control (64.6 ± 8.2 vs. 84.0 ± 9.9). 52 NF4-expressing and 80 Vector control myotubes were used to measure the resting cytosolic Ca^{2+} levels. *Significant difference compared with Vector control ($p < 0.05$).

which are known to be responsible for extracellular Ca^{2+} entries in skeletal myotubes [4], the expression levels of Orai1 and its regulator, STIM1, were examined by immunoblot analysis (Fig. 4C). No changes in the expression levels of either Orai1 or STIM1 were caused by the NF4 expression. Therefore, these results suggest that the inhibition of the heteromeric TRPC3/1 formation by the very end sub-region of TRPC3 resulted in a decrease in the resting cytosolic Ca^{2+} levels of the skeletal myotubes.

Considering that the change in cytosolic Ca^{2+} levels is one of the key intracellular events evoking muscle contraction and relaxation, which are the main task of skeletal muscle [4,5], the maintenance of a certain degree of resting cytosolic Ca^{2+} levels is an important requisite for keeping skeletal myotubes at rest, and the contribution of heteromeric TRPC3/1 to the maintenance of the resting cytosolic Ca^{2+} levels of skeletal muscle was found in the present study.

3.6. Perspectives

Patients in the pre-symptomatic phase of DMD show altered expressions of more than 30 proteins containing AR [27], and the involvement of TRP channels in an abnormal Ca^{2+} elevation in the subcellular space of skeletal myotubes from DMD patients has been strongly suggested [28]. The present study revealed the role of the AR region in heteromeric TRPC3/1 formation and the role of heteromeric TRPC3/1 in the maintenance of the cytosolic Ca^{2+} levels of skeletal muscle. Therefore, AR-containing TRPC3 could be a useful target for investigating novel diagnoses and treatments for skeletal muscle diseases such as DMD [29].

Acknowledgments

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Appendix A. Supplementary data

Supplementary data associated with this article can be found, in the online version, at http://dx.doi.org/10.1016/j.bbrc.2014.02.127.

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