Biochemical and Biophysical Research Communications 394 (2010) 126–129

Contents lists available at ScienceDirect

Biochemical and Biophysical Research Communications

journal homepage: www.elsevier.com/locate/ybbrc

A new property of twitchin to restrict the "rolling" of mussel tropomyosin and decrease its affinity for actin during the actomyosin ATPase cycle

Stanislava V. Avrova [a], Nikolay S. Shelud'ko [b], Yurii S. Borovikov [a,*]

[a] Laboratory of Mechanisms of Cell Motility, Institute of Cytology, Russian Academy of Sciences, 4 Tikhoretsky Avenue, St. Petersburg 194064, Russia
[b] Laboratory of Cell Biophysics, Institute of Marine Biology Far East Branch of the RAS, Vladivostok, Russia

ARTICLE INFO

Article history:
Received 10 February 2010
Available online 23 February 2010

Keywords:
Mollusc catch muscle
Twitchin
Ghost muscle fibers
ATPase cycle
Actomyosin intermediate states
Polarized fluorescence

ABSTRACT

A new evidence on the regulatory function of twitchin, a titin-like protein of molluscan muscles, at muscle contraction has been obtained at studying the movements of IAF-labeled mussel tropomyosin in skeletal ghost fibers during the ATP hydrolysis cycle simulated using nucleotides and non-hydrolysable ATP analogs. For the first time, myosin-induced multistep changes in mobility and in the position of mussel tropomyosin strands on the surface of the thin filament during the ATP hydrolysis cycle have been demonstrated directly. Unphosphorylated twitchin shifts the tropomyosin towards the position typical for muscle relaxation, decreases the tropomyosin affinity to actin and inhibits its movements during the ATPase cycle. Phosphorylation of twitchin by the catalytic subunit of protein kinase A reverses this effect. These data imply that twitchin is a thin filament regulator that controls actin–myosin interaction by "freezing" tropomyosin in the blocked position, resulting in the inhibition of the transformation of weak-binding states into strong-binding ones during ATPase cycle.

1. Introduction

Muscle contraction is generated by the interaction of myosin cross-bridges with actin filaments and ATP. During force generation, the myosin cross-bridges pass through several conformational states, the so-called "strong" and "weak" forms of myosin binding to actin [1,2]. In all muscle types, a constituent of thin filaments is tropomyosin. Its polymers formed by coiled-coil dimers joined end-to-end, are located in the grooves of the F-actin double helix. It is generally accepted that the movement of tropomyosin polymers on actin filaments plays an important role in the thin filament-based regulatory process of muscle contraction [1]. In the muscles that have only thin filament-based Ca^{2+}-regulation (striated skeletal and cardiac muscles of vertebrates), the interaction of myosin with actin is regulated by the movements of the tropomyosin–troponin complex, located on actin filaments, in response to a change in intracellular [Ca^{2+}] concentration. Both structural and biochemical data suggest that tropomyosin (TM) strands can occupy three different positions on actin ("blocked" or calcium-free, "closed" or calcium-induced and myosin-induced or "open"), depending on the presence or absence of troponin (TN), myosin, and Ca^{2+} [1–3]. It is suggested that in the "blocked" position, TM strands block the specific myosin-binding site on the actin filament, resulting in the inhibition of actin–myosin interaction. The binding of Ca^{2+} to TN eliminates this inhibition by azimuthal movement of TM strands, allowing myosin to interact freely with actin [4]. The strong binding of the myosin heads with actin shifts TM strands further towards the center of the thin filament, to the "open" position [5].

The primary regulatory system in molluscan catch muscles is myosin-linked and operates through Ca^{2+} binding to the essential light chains on the myosin heads [6]. In addition, there likely to be the actin-linked regulatory system involving TM and caldesmon [7] or calponin [8] or TN [9]. It is known that some molluscan smooth muscles possess a "catch" state, a unique capability to maintain tension and particular resistance to stretch for long time periods with low energy consumption [10,11]. The catch state is explained by the formation of cross-linkages between filaments within the contractile apparatus [12]. Although the molecular mechanism of the catch state is still unknown [10,11], it is well established that release of the catch requires phosphorylation of thick filament-associated titin-like protein twitchin [13] by cAMP-dependent protein kinase (PKA) [14].

According to this view, the catch linkages are formed as a result of twitchin dephosphorylation by a Ca^{2+}-stimulated phosphatase [15], and remain in the locked state when Ca^{2+} is decreased to basal

Abbreviations: S1, myosin subfragment-1; TM, tropomyosin; TN, troponin complex; IAF, 5-iodoacetamido fluorescein; DTT, dithiothreitol; PKA, cAMP-dependent protein kinase; EGTA, ethylene glycol-bis (2-aminoethylether)-*N*,*N*,*N*′,*N*′-tetraacetic acid

* Corresponding author. Address: Laboratory of Mechanisms of Cell Motility, Institute of Cytology, Russian Academy of Sciences, 4 Tikhoretsky Avenue, St. Petersburg 194064, Russia. Fax: +7 812 297 0341.
E-mail address: boroviko@mail.cytspb.rssi.ru (Y.S. Borovikov).

0006-291X/$ - see front matter © 2010 Elsevier Inc. All rights reserved.
doi:10.1016/j.bbrc.2010.02.128

levels after cessation of stimulation. The cholinergic excitation of catch muscle causes a transient increase in the intracellular Ca^{2+} that activates thin and thick filaments and twitchin phosphatase. As a result, two processes start in parallel: the active muscle contraction and the formation of twitchin linkages. If the catch linkages were already formed during active contraction, they would prohibit an action of force-generating myosin bridges. However, some experimental data suggest that muscle shortening is not prevented when the catch linkages are formed [13,16]. The inverse relationship between catch force and active force suggests that myosin cross-bridges in the strong-binding state make the catch linkages to detach, whereas myosin in the weak-binding state promotes their formation [17]. It was explained by assuming that twitchin and the myosin heads compete for a common binding site on actin [17,18]. Another assumption is that the mechanism of the interdependent influence of active and passive bridges on each other involves the regulating effect of tropomyosin on the actin–myosin interaction [19].

It was found that twitchin could specifically interact not only with molluscan contractile proteins but also with actin and myosin from rabbit skeletal muscle [18–20] and inhibit actin-activated Mg^{2+}-ATPase of skeletal actomyosin [18–20]. Addition of twitchin induces a catch-like stiffness in skinned skeletal muscle fibers [21,22]. The skinned and ghost fibers prepared from skeletal muscle fibers were shown to be fit for testing some properties of that protein [23,24].

Here, we used the model system of reconstructed skeletal ghost muscle fibers and polarization fluorimetry to study the effect of twitchin on the mobility and movement of mussel tropomyosin at various intermediate stages of the ATP hydrolysis cycle. The nucleotide-dependent conformational changes of fluorescently labeled TM were monitored in the absence of nucleotides and in the presence of either MgADP, MgAMP-PNP, MgATPγS or MgATP. The conformational changes in TM during the ATPase cycle, manifested in multistep changes in mobility and spatial arrangement of tropomyosin strands, were observed. Unphosphorylated twitchin, upon its addition to the ghost fibers, increased the motility of TM (decreased TM affinity for actin) and inhibited the shifting or "rolling" of tropomyosin, "freezing" TM in position typical for relaxation muscle fibers. The addition of PKA catalytic subunit reverses the effect of twitchin. These data demonstrated that twitchin in a phosphorylation-dependent manner could control the conformational state of TM, its affinity for actin and the position of TM strands on the thin filament.

2. Materials and methods

Twitchin and tropomyosin were prepared from the posterior adductor of the mussel *Crenomytilus grayanus* as it was described [19,20]. Cys135 of TM [25] was labeled with 5-iodoacetamide fluorescein (IAF) as described by Lamkin et al. [26], producing a probe to protein molar ratio 0.8:1. The modification of Cys135 does not significantly affect the functional properties of TM. Human fast skeletal troponin subunits were expressed in BL21(DE3) *Escherichia coli* and purified as previously reported [27]. Myosin subfragment-1 (S1) devoid of regulatory light chains was prepared by treatment of skeletal muscle myosin with α-chymotrypsin [28]. The purity of all proteins was verified by polyacrylamide gel electrophoresis in the presence of sodium dodecylsulfate (SDS–PAGE).

Glycerinated muscle fibers were obtained from rabbit *psoas* muscles by the method of Szent-Gyorgyi [29]. Ghost fibers were prepared from single glycerinated fibers as described earlier [30]. Binding of S1, tropomyosin and twitchin to F-actin was carried out by incubation of each of the proteins (2–3 mg/ml) with the fibers in a solution containing 20 mM KCl, 3 mM $MgCl_2$, 1 mM DDT, and 10 mM Tris–HCl (pH 6.8) (buffer A) as described previously [31]. The unbound proteins were washed out by the incubation of the fibers in the buffer A for 15 min at room temperature. In some experiments 85 U ml^{-1} PKA from bovine heart (Sigma P-2645) were added in buffer A. The effectiveness of the reconstruction of the filaments in the ghost muscle fibers used for fluorescent measurements was verified by examining the protein content by SDS–PAGE with subsequent densitometry of the gels (UltroScan XL, Pharmacia LKB). The fibers in the final preparations contained actin, S1, tropomyosin, twitchin and Z-line proteins. The molar ratios of tropomyosin, S1 and twitchin to actin in the ghost fibers were approximately 1:6.5 (±2), 1:5 (±2), and 1:14 (±2), respectively.

Steady-state fluorescence polarization measurements on single ghost muscle fibers were made using a photometer [31]. The polarized fluorescence from IAEDANS-labeled S1 was recorded at 500–600 nm after excitation at 437 ± 5 nm. The measurements were carried out in the buffer A in the absence of nucleotides or in the presence of either 2.5 mM ADP, 25 mM AMP-PNP, 15 mM ATPγS or 5 mM ATP. In the absence of nucleotides, the AM state of the actomyosin complex was simulated. MgADP, MgAMP-PNP, MgATPγS, and MgATP were used for mimicking intermediate states of actomyosin, AM^·ADP, AM′·ADP, AM*·ATP, and AM**·ADP·Pi, respectively [32,33], where A is actin and M, M^, M′, M*, and M** are various conformational states of the myosin head. We used solutions with a lower ionic strength than that in situ, in order to increase the affinity of myosin S1 to actin at the simulated weak-binding states. In our experiments, we observed the retention of the functional properties of the contractile and regulatory proteins in muscle fibers even at a low ionic strength, if the solutions contained 3 mM Mg^{2+} [3].

The intensities of the four components of polarized fluorescence ${}_{||}I_{||}$, ${}_{||}I_{\perp}$, ${}_{\perp}I_{\perp}$, and ${}_{\perp}I_{||}$ were measured using a photometer [31]. The subscripts || and ⊥ designate the orientation of polarization plane parallel or perpendicular to the fiber axis, the former denoting the direction of polarization of the incident light and the latter – that of the emitted light. Fluorescence polarization ratios were defined as: $P_{||} = ({}_{||}I_{||} - {}_{||}I_{\perp})/({}_{||}I_{||} + {}_{||}I_{\perp})$ and $P_{\perp} = ({}_{\perp}I_{\perp} - {}_{\perp}I_{||})/({}_{\perp}I_{\perp} + {}_{\perp}I_{||})$. Parameters $P_{||}$ and $P_{\perp}$ were used to evaluate the anisotropy of polarized fluorescence of the fiber.

To estimate the changes in the probe orientation we used the model-dependent method [31,34]. The following assumptions were made: in the fiber, the fluorescent probes have either an ordered (fraction N) or disordered (fraction $1 - N$) orientation; the absorption and emission of light is accomplished by linear, completely anisotropic dipoles of absorption ($\vec{A}$) and emission ($\vec{E}$); the axes of the dipoles of the orderly oriented probes are arranged in a spiral along the surface of the cone, whose axis coincides with the long axis of the thin filament; the dipoles of absorption and emission form the angles Φ_A and Φ_E, respectively, at the top of the cone. In terms of this model, the changes in Φ_A, Φ_E, and N are considered to reflect changes in orientation and mobility of the protein containing the probe.

Since in all the experiments the values of Φ_E and Φ_A were found to be very close, only Φ_E values were presented in the figure. The statistical reliability of the changes was evaluated using Student's t-test. The changes of the parameters were statistically significant ($p < 0.05$).

3. Results and discussion

In agreement with earlier findings [35], the binding of AF-TM to F-actin of the ghost fibers resulted in polarized fluorescence, with $P_{||}$ values being higher than $P_{\perp}$ (Table 1), which indicated that the probe dipoles were predominantly oriented parallel to the fiber

Table 1
The effect of S1, TN, the nucleotides and twitchin (TW) on polarization ratios of IAF bound to Cys135 of tropomyosin in the ghost fibers.

Nucleotide	TN	TW	S1	*n*	$P_{\|\|}$ ± SEM	$P_{\perp}$ ± SEM
None	–	–	–	7	0.183 ± 0.001	0.124 ± 0.002
	+	–	–	6	0.168 ± 0.001	0.114 ± 0.002
	+*	–	–	6	0.194 ± 0.001	0.137 ± 0.002
	–	+	–	8	0.153 ± 0.002	0.114 ± 0.002
	–	–	+	9	0.181 ± 0.001	0.069 ± 0.002
	–	+	+	5	0.166 ± 0.002	0.105 ± 0.002
ADP	–	–	+	5	0.196 ± 0.002	0.072 ± 0.002
	–	+	+	5	0.175 ± 0.002	0.122 ± 0.002
AMP-PNP	–	–	+	5	0.201 ± 0.002	0.105 ± 0.002
	–	+	+	5	0.179 ± 0.002	0.135 ± 0.003
ATPγS	–	–	+	5	0.127 ± 0.002	0.114 ± 0.003
	–	+	+	5	0.131 ± 0.002	0.129 ± 0.003
ATP	–	–	+	5	0.126 ± 0.002	0.116 ± 0.003
	–	+	+	6	0.134 ± 0.002	0.137 ± 0.003

$P_{||}$ and $P_{\perp}$ were calculated as described in Section 2. *n* is the number of fibers used in the experiments. S1, TN, TW, the nucleotides and Ca^{2+} have a pronounced effect on the values of $P_{||}$ and $P_{\perp}$, indicating the changes in the conformational state of TM in the ghost fibers ($p < 0.05$). Error indicates ± SEM. +* show TN at high-Ca^{2+}.

axis. The model-dependent method revealed the angle Φ_E to be close to 52° (Fig. 1A). The relative amount of disorderly oriented probes (*N*) did not exceed 0.3. Taking into account the rigid binding of the probe to the proteins, that suggested a highly-ordered arrangement of TM in the fibers (for a review, see [36]).

According to the data presented in Fig. 1A binding of TN at high-Ca^{2+} or binding of S1 to F-actin decorated by AF-TM decrease the values of Φ_E, showing the tilting of the probe to the fiber axis. In contrast, the addition of either TN at low-Ca^{2+} or twitchin induces the increase in Φ_E values, showing the tilting of the probe away from the fiber axis. There is growing evidence in support of the suggestion that TN and S1 can cause a shift or "rolling" [37] of TM strands across the thin filament surface in any type of muscles [38,39,4]. It is possible that the decrease in the values of Φ_E observed at binding of S1 or TN at high-Ca^{2+} to actin indicates a shift or "rolling" of mussel TM strands towards the center of the thin filament, to the open position. In contrast, the addition of TN at low-Ca^{2+} in the absence of S1 or the addition of twitchin both in the absence and presence of S1 increases Φ_E values showing the movement of TM strands towards the periphery of the thin filament to the blocked position. It is interesting, that the addition of PKA catalytic subunit reverses the effect of twitchin (Fig. 1). This means that only unphosphorylated twitchin induces the conformational changes in TM, resulting in a shifting or "rolling" of TM strands towards the blocked position on the thin filament. Unphosphorylated twitchin decreases also the affinity of mussel TM binding to actin in thin filaments as follows from the increase in the *N* value (Fig. 1B) [35].

Since there are no data on whether twitchin can bind to TM, but it has been shown to bind to actin [18–20], we suggest that twitchin binds to actin in the ghost fibers and this binding induces the shift of TM strands towards the blocked position and decreases the affinity of TM binding to actin. Similar correlation between the changes in Φ_E values and the character of the movement of TM strands across the thin filament surface has also been revealed in our previous studies on the mechanisms of the ATPase cycle regulation by skeletal and smooth muscle tropomyosins [35,40]. It is possible that twitchin in a phosphorylation-dependent manner can control the conformational state of TM, its affinity for actin and the position of TM strands on the thin filament.

In the presence of S1, the position of TM on the thin filaments and their affinity for actin change in a nucleotide-dependent manner. At transition from AM**·ADP·Pi to AM state the values of *N* and Φ_E decrease (Fig. 1). Unphosphorylated twitchin markedly inhibits this effect. Since the decrease and increase of the angle Φ_E correlates with the movement of TM strands to the center and to the

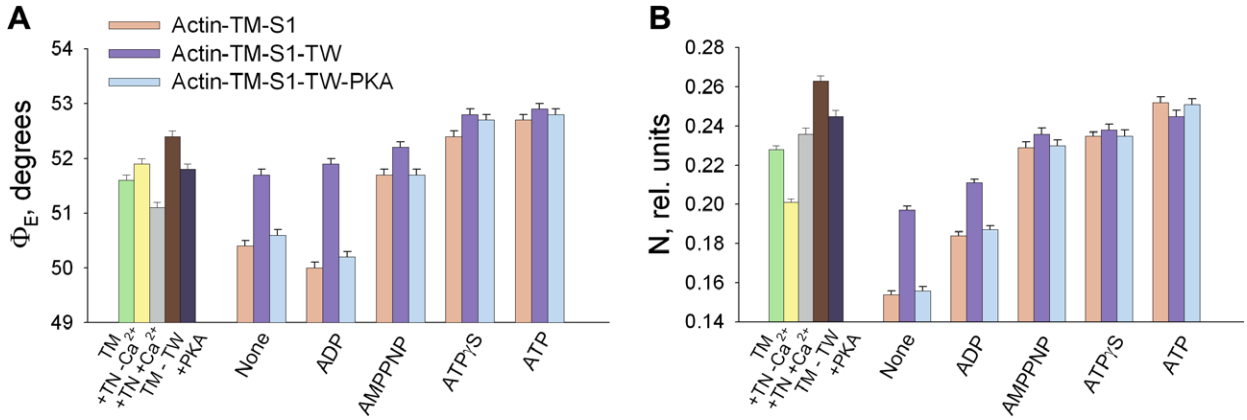

Fig. 1. The effect of twitchin (TW), troponin (TN) and Ca^{2+} on the values of Φ_E (A) and *N* (B) of the polarized fluorescence of AF-TM revealed in ghost fibers at simulating the sequential steps of the ATPase cycle. Φ_E is the angle between the emission dipole of the probe and the thin filament axis; *N* is a number of disorderly oriented fluorophores. Calculations of the values of Φ_E and *N*, the preparation of the fibers, their composition, and the conditions of the experiments are described in Materials and methods. The data represent means of 5–6 ghost fibers for each experimental condition (see Table 1). The Φ_E and *N* values in the absence and in the presence of the nucleotides are significantly altered by S1 and twitchin ($p < 0.05$). Error bars indicate ±SEM.

periphery of the thin filament, respectively [4,35,40], this suggests that during the ATPase cycle S1 shifts TM strands to the thin filament center towards the open position, whereas twitchin shifts it towards the blocked position under the same conditions. Consequently, twitchin reverses the expected movement of mussel TM (as observed in the absence of twitchin) to the center of the thin filament. Since each intermediate state of actomyosin corresponds to a definite conformational state and position of TM on the thin filament [35], it is possible to suggest that the reverse of the motion of TM strands towards the blocked position (Fig. 1A) can inhibit the formation of the strong-binding states during ATP cycle. It is quite likely that in the catch state twitchin can maintain a low level of energy utilization by "freezing" tropomyosin strands in the position typical for fiber relaxation, i.e. in the states at which actin–myosin interaction is inhibited.

The experimental approach used in this work as well as in some previous studies [35,40] allowed us to follow the rearrangement of the proteins (F-actin and tropomyosin) in the thin filament under the influence of the myosin heads during the ATPase cycle. The present work has shown that twitchin can alter the position of tropomyosin in thin filaments reconstituted in ghost fibers and thereby affect the ability of myosin heads to move tropomyosin. The direct evidences in support of caldesmon and troponin-induced tropomyosin rolling in smooth and skeletal muscle thin filaments have been presented [41]; these data argue in favor of an active role of caldesmon and troponin in positioning the tropomyosin molecule. It is quite possible that in catch muscle, the similar role in tropomyosin positioning is played by twitchin, and our data support its role in this process.

Acknowledgments

The authors thank Dr. Charles S. Redwood and Dr. Paul Robinson for their gift of skeletal troponin. This work was supported by the RFFR (08-04-00960a and 08-04-00793a), MCB program of the RAS and "The Leading Scientific Schools of Russia" program (NSH-4132.2010.4).

References

[1] S.V. Perry, Vertebrate tropomyosin: distribution, properties and function, J. Muscle Res. Cell Motil. 22 (2001) 5–49.
[2] M.A. Geeves, K.C. Holmes, The molecular mechanism of muscle contraction, Adv. Protein Chem. 71 (2005) 161–193.
[3] R. Maytum, S.S. Lehrer, M.A. Geeves, Cooperativity and switching within the three-state model of muscle regulation, Biochemistry 38 (1999) 1102–1110.
[4] W. Lehman, R. Craig, P. Vibert, Ca^{2+}-Induced tropomyosin movement in *Limulus* thin filaments revealed by three-dimensional reconstruction, Nature 368 (1994) 65–67.
[5] R. Craig, W. Lehman, Crossbridge and tropomyosin positions observed in native, interacting thick and thin filaments, J. Mol. Biol. 311 (2001) 1027–1036.
[6] A.G. Szent-Gyorgyi, Regulation by myosin: how calcium regulates some myosins, past and present, in: S. Ebashi, I. Ohtsuki (Eds.), Regulatory Mechanisms of Striated Muscle Contraction, Springer, Tokyo, 2007, pp. 253–264.
[7] P.M. Bennett, S.B. Marston, Calcium regulated thin filaments from molluscan catch muscles contain a caldesmon-like regulatory protein, J. Muscle Res. Cell Motil. 11 (1990) 302–312.
[8] D. Funabara, M. Nakaya, S. Watabe, Isolation and characterization of a novel 45 kDa calponin-like protein from anterior byssus retractor muscle of the mussel *Mytilus galloprovincialis*, Fish Sci. 67 (2001) 511–517.
[9] K. Nishita, T. Ojima, A. Takahashi, A. Inoue, Troponin from smooth adductor muscle of Ezo-giant scallop, J. Biochem. 121 (1997) 419–424.
[10] D. Funabara, S. Kanoh, M.J. Siegman, T.M. Butler, D.J. Hartshorne, S. Watabe, Twitchin as a regulator of catch contraction in molluscan smooth muscle, J. Muscle Res. Cell Motil. 26 (2005) 455–460.
[11] S. Galler, Molecular basis of the catch state in molluscan smooth muscles: a catchy challenge, J. Muscle Res. Cell Motil. 29 (2008) 73–99.
[12] J.C. Ruegg, Smooth muscle tone, Physiol. Rev. 51 (1971) 201–248.
[13] T.M. Butler, S.U. Mooers, C. Li, S. Narayan, M.J. Siegman, Regulation of catch muscle by twitchin phosphorylation: effects on force, ATPase, and shortening, Biophys. J. 75 (1998) 1904–1914.
[14] R.K. Achazi, B. Dolling, R. Haakshorst, 5-HT-induced relaxation and cyclic AMP in a molluscan smooth muscle, Pflugers Arch. 349 (1974) 19–27.
[15] L. Castellani, C.A. Cohen, Calcineurin-like phosphatase is required for catch contraction, FEBS Lett. 309 (1992) 321–326.
[16] M. Mukou, H. Kishi, I. Shirakawa, T. Kobayashi, K. Tominaga, H. Imanishi, H. Sugi, Marked load-bearing ability of *Mytilus* smooth muscle in both active and catch states as revealed by quick increases in load, J. Exp. Biol. 207 (2004) 1675–1681.
[17] T.M. Butler, S.U. Mooers, M.J. Siegman, Catch force links and the low to high force transition of myosin, Biophys. J. 90 (2006) 3193–3202.
[18] D. Funabara, C. Hamamoto, K. Yamamoto, A. Inoue, M. Ueda, R. Osawa, S. Kanoh, D.J. Hartshorne, S. Suzuki, S. Watabe, Unphosphorylated twitchin forms a complex with actin and myosin that may contribute to tension maintenance in catch, J. Exp. Biol. 210 (2007) 4399–4410.
[19] N.S. Shelud'ko, O.S. Matusovsky, T.V. Permyakova, G.G. Matusovskaya, "Twitchin-actin linkage hypothesis" for the catch mechanism in molluscan muscles: evidence that twitchin interacts with myosin, myorod, and paramyosin core and affects properties of actomyosin, Arch. Biochem. Biophys. 466 (2007) 125–135.
[20] N.S. Shelud'ko, G.G. Matusovskaya, T.V. Permyakova, O.S. Matusovsky, Twitchin, a thick-filament protein from molluscan catch muscle, interacts with F-actin in a phosphorylation-dependent way, Arch. Biochem. Biophys. 432 (2004) 269–277.
[21] S.V. Avrova, N.S. Shelud'ko, Yu.S. Borovikov, S. Galler, Twitchin of mollusk muscle increases stiffness of skinned human skeletal muscle fibres, J. Muscle Res. Cell Motil. 29 (2008) 289.
[22] S.V. Avrova, N.S. Shelud'ko, Yu.S. Borovikov, S. Galler, Twitchin of mollusc smooth muscles can induce "catch"-like properties in human skeletal muscle: support for the assumption that the "catch" state involves twitchin linkages, J. Comp. Physiol. B 179 (2009) 945–950.
[23] Yu.S. Borovikov, N.S. Shelud'ko, S.V. Avrova, Molluscan twitchin can control actin–myosin interaction during ATPase cycle, Arch. Biochem. Biophys. (2010), doi:10.1016/j.abb.2010.01.001.
[24] N.A. Rysev, S.V. Avrova, L.A. Solov'eva, N.S. Shelud'ko, Yu.S. Borovikov, The effect of twitchin on intermediate actomyosin states in glycerol-skinned skeletal muscle fibres, J. Muscle Res. Cell Motil. 29 (2008) 290.
[25] K. Iwasaki, K. Kikuchi, D. Funabara, S. Watabe, CDNA cloning of tropomyosin from the anterior byssus retractor muscle of mussel and its structural integrity from the deduced amino acid sequence, Fish Sci. 63 (1997) 731–734.
[26] M. Lamkin, T. Tao, S.S. Lehrer, Tropomyosin–troponin and tropomyosin–actin interactions: a fluorescence quenching study, Biochemistry 22 (1983) 3053–3058.
[27] P. Robinson, S. Lipscomb, L.C. Preston, E. Altin, H. Watkins, C.C. Ashley, C.S. Redwood, Mutations in fast skeletal troponin I, troponin T, and beta-tropomyosin that cause distal arthrogryposis all increase contractile function, FASEB J. 21 (2007) 896–905.
[28] Y. Okamoto, T. Sekine, A streamlined method of subfragment one preparation from myosin, J. Biochem. (Tokyo) 98 (1985) 1143–1145.
[29] A.G. Szent-Gyorgyi, Meromyosins, the subunits of myosin, Arch. Biochem. Biophys. 42 (1953) 305–320.
[30] Y.S. Borovikov, N.B. Gusev, Effect of troponin-tropomyosin complex and Ca^{2+} on conformational changes in F-actin induced by myosin subfragment-1, Eur. J. Biochem. 136 (1983) 363–369.
[31] Y.S. Borovikov, I.V. Dedova, C.G. dos Remedios, N.N. Vikhoreva, P.G. Vikhorev, S.V. Avrova, T.L. Hazlett, B.W. Van Der Meer, Fluorescence depolarization of actin filaments in reconstructed myofibres: the effect of S1 or pPDM-S1 on movements of distinct areas of actin, Biophys. J. 86 (2004) 3020–3029.
[32] R.S. Goody, W. Hofmann, Stereochemical aspects of the interaction of myosin and actomyosin with nucleotides, J. Muscle Res. Cell Motil. 1 (1980) 101–115.
[33] O. Roopnarine, D.D. Thomas, Orientation of intermediate nucleotide states of indane dione spin-labeled myosin heads in muscle fibres, Biophys. J. 70 (1996) 2795–2806.
[34] G.W. Tregear, R.A. Mendelson, Polarization from a helix of fluorophores and its relation to that obtained from muscle, Biophys. J. 15 (1975) 455–467.
[35] Y.S. Borovikov, O.E. Karpicheva, S.V. Avrova, C.S. Redwood, Modulation of the effects of tropomyosin on actin and myosin conformational changes by troponin and Ca^{2+}, Biochim. Biophys. Acta 1794 (2009) 985–994.
[36] Y.S. Borovikov, Conformational changes of contractile proteins and their role in muscle contraction, Int. Rev. Cytol. 189 (1999) 267–301.
[37] L.M.F. Holthauzen, F. Corrêa, C.S. Farah, Ca^{2+}-induced rolling of tropomyosin in muscle thin filaments, J. Biol. Chem. 279 (2004) 15204–15213.
[38] K.J. Poole, M. Lorenz, G. Evans, G. Rosenbaum, A. Pirani, R. Craig, et al., A comparison of muscle thin filament models obtained from electron microscopy reconstructions and low-angle X-ray fibre diagrams from non-overlap muscle, J. Struct. Biol. 155 (2006) 273–284.
[39] H.S. Jung, R. Craig, Ca^{2+}-Induced tropomyosin movement in scallop striated muscle thin filaments, J. Mol. Biol. 383 (2008) 512–519.
[40] N. Kulikova, O.E. Pronina, R. Dabrowska, Y.S. Borovikov, Caldesmon restricts the movement of both C- and N-termini of tropomyosin on F-actin in ghost fibres during the actomyosin ATPase cycle, Biochem. Biophys. Res. Commun. 345 (2006) 280–286.
[41] C. Bacchiocchi, P. Graceffa, S.S. Lehrer, Myosin-induced movement of alphaalpha, alphabeta, and betabeta smooth muscle tropomyosin on actin observed by multisite FRET, Biophys. J. 86 (2004) 2295–2307.