Available online at www.sciencedirect.com
SCIENCE DIRECT®
Biochemical and Biophysical Research Communications 337 (2005) 195–200
BBRC
www.elsevier.com/locate/ybbrc

Selective expression of the type 3 isoform of ryanodine receptor Ca^{2+} release channel (RyR3) in a subset of slow fibers in diaphragm and cephalic muscles of adult rabbits

Antonio Conti [a], Carlo Reggiani [b], Vincenzo Sorrentino [c,*]

[a] *DIBIT, San Raffaele Scientific Institute, Milan, Italy*
[b] *Department of Anatomy and Physiology, University of Padova, Italy*
[c] *Molecular Medicine Section, Department of Neuroscience, University of Siena, 53100 Siena, Italy*

Received 31 August 2005
Available online 15 September 2005

Abstract

The expression pattern of the RyR3 isoform of Ca^{2+} release channels was analysed by Western blot in neonatal and adult rabbit skeletal muscles. The results obtained show that the expression of the RyR3 isoform is developmentally regulated. In fact, RyR3 expression was detected in all muscles analysed at 2 and 15 days after birth while, in adult animals, it was restricted to a subset of muscles that includes diaphragm, masseter, pterygoideus, digastricus, and tongue. Interestingly, all of these muscles share a common embryonic origin being derived from the somitomeres or from the cephalic region of the embryo. Immunofluorescence analysis of rabbit skeletal muscle cross-sections showed that RyR3 staining was detected in all fibers of neonatal muscles. In contrast, in those adult muscles expressing RyR3 only a fraction of fibers was labelled. Staining of these muscles with antibodies against fast and slow myosins revealed a close correlation between expression of RyR3 and fibers expressing slow myosin isoform.

Keywords: Ryanodine receptor; Muscle fiber types; Muscle development; Head and neck muscles; Calcium release channels

Muscle contraction requires the coordinated activation of the dihydropyridine receptors (DHPRs), the voltage sensors located on the plasma membrane and of the ryanodine receptor Ca^{2+} release channels on the sarcoplasmic reticulum. Three different isoforms of ryanodine receptor Ca^{2+} release channels are expressed in mammalian tissues [1,2]. The skeletal muscle channels (RyR1) and the cardiac channels (RyR2) are essential for excitation–contraction coupling in skeletal and cardiac muscle, respectively. In skeletal muscle, a direct coupling model between DHPR and RyR1 has been proposed. According to this model, RyR1 channels are physically coupled with DHPRs and open in relation to conformational changes of the DHPRs induced by membrane depolarization [1–4]. In the past years, we have reported that RyR3 channels are also expressed in mammalian skeletal muscles [5–7], where they represent about 1% of total RyR channels [8]. Since all available evidence indicates that only RyR1 channels are able to couple with DHPR [3,9,10], the role of the limited amounts of RyR3 present in mammalian skeletal muscles is not understood. Evidence of a possible involvement of RyR3 channels in regulating skeletal muscle contraction has been obtained following the observation that RyR3 channels are mostly expressed in the neonatal period of mammalian skeletal muscle development, when RyR3 is expressed in all skeletal muscles [11]. In fact, at variance with RyR1, which reaches the highest level of expression in adult life, expression of RyR3 increases from the last prenatal days, peaks between 7 and 15 days after birth, and is below detection levels in adult skeletal muscles [11,12]. The exception to this rule is the diaphragm muscle, where small amounts of RyR3 isoform can still be detected in adult animals [7]. In agreement with the wide expression

* Corresponding author. Fax: +39 0577 234 191.
E-mail address: v.sorrentino@unisi.it (V. Sorrentino).

0006-291X/$ - see front matter © 2005 Elsevier Inc. All rights reserved.
doi:10.1016/j.bbrc.2005.09.027

of RyR3 in embryonic and neonatal muscles, studies on the contractile properties of mice knockout for the RyR3 gene revealed that the amount of force generated upon electrical stimulation or following caffeine exposure is strongly depressed in skeletal muscles from newborn RyR3 −/− mice compared to normal controls [11]. These data suggest that, at least during post-natal development, RyR3 may participate in regulation of muscle contraction.

In order to extend our knowledge on RyR3 channels in mammalian skeletal muscles, we performed a careful analysis of the expression of RyR3 channels in adult animals by Western blot analysis and immunostaining experiments using RyR isoform-specific antibodies. We decided to perform our analysis on rabbit skeletal muscles, as this animal model offers the possibility to dissect more easily specific muscles and allowed us to extend our studies on expression of the RyR3 isoform to another mammalian species in addition to the mouse that was the subject of our previous work.

Previous analysis performed in mouse skeletal muscles had revealed that, in adult diaphragm, RyR3 expression is restricted to a small population (about 15%) of muscle fibers [12]. Experiments of caffeine-induced calcium release in diaphragm single fibers also showed that a subset of fibers showed a high responsiveness, which might be related to the presence of RyR3 [13]. In order to investigate whether RyR3 expression is associated with a specific fiber type in skeletal muscles, we examined the expression pattern of different myosin isoforms in RyR3-expressing fibers. Western blot analysis performed on different adult muscles showed that, in addition to diaphragm, RyR3 channels are selectively expressed in muscles of cephalic origin. In these muscles, expression of RyR3 was observed only in slow fibers.

Materials and methods

Animals. The study was carried out on newborn (2 and 15 days old) and adult (120 days old) New Zealand male rabbits, on adult CD1 mice (2 months old), and on adult Wistar rats (2 months old). Single muscles were dissected and either used for microsomal preparation or for immunohistochemistry. The study was approved by the Ethics Committee of the Department of Anatomy and Physiology, University of Padova.

Microsomal vesicle preparation and Western blot analysis. Microsomal fractions were prepared from rabbit skeletal muscles as previously described [7]. Muscles were homogenized in ice-cold buffer A (320 mM sucrose, 5 mM Na-Hepes, pH 7.4, and 0.1 mM PMSF) using a Dounce homogenizer. Homogenates were centrifuged at 7000*g* for 5 min at 4 °C. The supernatant obtained was centrifuged at 100,000*g* for 1 h at 4 °C. The microsomes were resuspended in buffer A and stored at −80 °C. Protein concentration of the microsomal fractions was quantified using the Bradford protein assay kit (Bio-Rad). Microsomal proteins were separated by SDS/PAGE and then transferred to a nitrocellulose membrane (Schleicher & Schuell). Membranes were incubated for 3 h in 150 mM NaCl, 50 mM Tris–HCl, pH 7.4, 0.2% Tween 20 plus 5% nonfat milk. Primary antibodies used were polyclonal rabbit antisera (diluted 1:3000) against the RyR isoforms. Preparation and isoform specificity of these antibodies have been described [5,6]. Biotinylated secondary antibody detection was performed using the amplified alkaline phosphatase detection method [5,6].

Immunohistochemistry. Skeletal muscles isolated from rabbits were frozen in cold 2-methylbutane and then in liquid nitrogen and stored at −80 °C. Ten micrometer sections were cut using a Leica cryostat and then mounted on glass microscope slides. Sections were either used immediately or stored at −80 °C before usage. Serial sections were stained with the following primary antibodies: anti-RyR1, anti-RyR3, BA-F8, and SC-71. The polyclonal antibodies anti-RyR1, anti-RyR3 are specific to the corresponding RyR isoforms as previously described [5,6,12]. The BA-F8 monoclonal antibody is specific to MHC-1 (also called β/slow) in rat and mouse [14–16], and may show cross-reactivity with MHC-α [17], whereas the monoclonal antibody SC-71 is specific for MHC-2A in rat and mouse [14–16], but reacts also with MHC-2X in rabbit [18]. For immunostaining, tissue sections were blocked for 2 h at 25 °C in PBS, 0.2% BSA 0.5% Triton, and 5% goat serum, and then incubated with the primary antibody overnight at 4 °C. The sections were subsequently washed with four changes of PBS/BSA/Triton for a total of 1 h. Sections were then incubated with fluorochrome-conjugated secondary antibody for 1 h at room temperature. The sections were washed three times and then mounted with Mowiol with 0.025% DABCO.

Results

RyR3 expression in adult rabbit skeletal muscles

The expression of the RyR1 and RyR3 isoforms of Ca^{2+} release channel was investigated in adult rabbit skeletal muscles by Western blot analysis. As shown in Fig. 1, immunodetection of RyR1 using an isoform-specific antibody revealed that RyR1 protein is expressed in all skeletal muscles of adult rabbits, although a certain degree of variation in the RyR1 can be observed among different muscles. Differences in the levels of expression of sarcoplasmic reticulum proteins, including RyR1, have been reported by other laboratories [19]. In agreement with previous results obtained in mice [5,6,11], analysis of RyR3 expression revealed that RyR3 channels could be clearly detected in adult diaphragm muscle of the rabbit. However, a clear RyR3-specific signal was also detected in other skeletal muscles including digastricus (anterior belly), pterygoideus (lateral), tongue, and masseter. No RyR3 signal was detected in EDL, tibialis anterior, gastrocnemius, psoas, vastus lateralis, longissimus, and soleus. It is interesting to note that in those muscles expressing RyR3, the levels of expression of RyR1 are apparently lower than those observed in muscles that do not express RyR3.

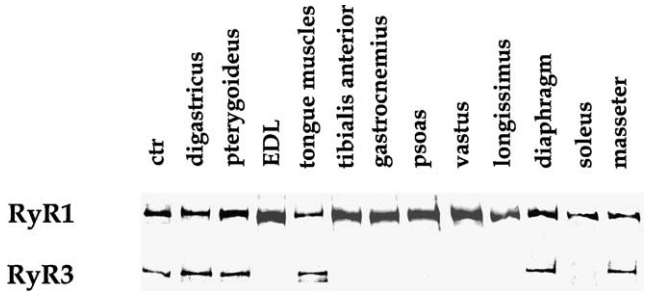

Fig. 1. Representative Western blots of adult rabbit skeletal muscles probed with anti-Ryr1 and anti-RyR3 antibodies. Each lane was loaded with 10 and 30 μg of microsomal proteins for identification of RyR1 and RyR3, respectively. Ctr, positive control.

RyR3 expression in developing rabbit skeletal muscle

As the expression of RyR3 in mouse skeletal muscles is known to be developmentally regulated [11], the analysis of RyR3 expression in rabbit was performed also on microsomes prepared from dissected skeletal muscle from 2, 15, and 120 day animals. As shown in Fig. 2, RyR3 protein was detected in all microsomal fractions prepared from skeletal muscles of 2- and 15-day-old rabbits. In agreement with data in Fig. 1, RyR3 was detected in diaphragm, digastricus, pterygoideus, tongue, and masseter muscles from 120-day-old rabbits. In order to extend and confirm these findings, we also analysed by Western blot the expression of RyR3 in skeletal muscles from adult mice and rats.

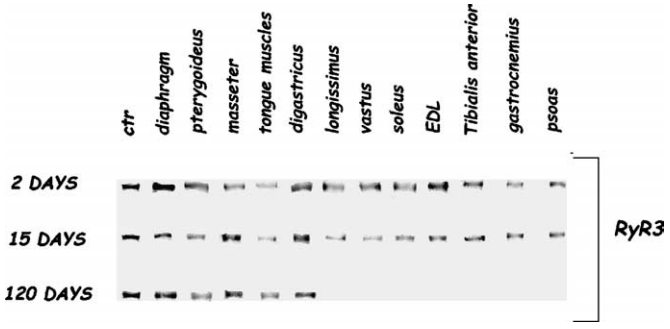

Fig. 2. Representative Western blot presenting the expression pattern of RyR3 in skeletal muscles of 2-, 15- or 120-day-old rabbits. Each lane was loaded with 30 μg, after transfer to nylon membranes, the blots were incubated with a polyclonal antibody against RyR3. Ctr, positive control.

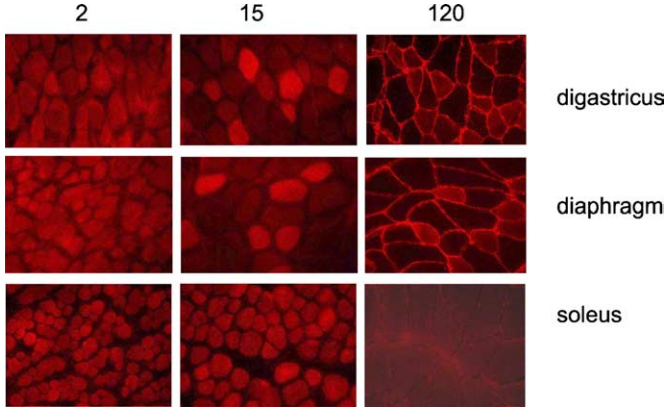

Fig. 3. Immunofluorescence of RyR3 expression in digastricus and diaphragm muscles of rabbit of 2, 15, and 120 days.

The results obtained confirmed the expression of RyR3 in masseter and digastricus muscles (data not shown), thus extending the data obtained in rabbits to these species. Expression of RyR3 was observed in tongue muscle of adult mice but not in adult rats (not shown).

Immunohistochemical analysis of RyR3 expression in developing rabbit skeletal muscles

Previous studies performed on cryosections obtained from murine skeletal muscles showed that, in neonatal muscles, RyR3 was not only expressed in all muscles analysed, but that it was also present in all fibers. In contrast, in adult mouse diaphragm RyR3 expression was retained only in less than 15% of the fibers [12]. To further characterize the distribution of RyR3 during skeletal muscle development in rabbit, cryosections of rabbit skeletal muscles of different ages were prepared and immunolabelled with specific antibodies against RyR3. In agreement with the results obtained by Western blot analysis, a specific staining for RyR3 was observed in all fibers of diaphragm, digastricus, and soleus muscles from 2-day-old rabbits (Fig. 3). In the same muscles from 15-day-old rabbits, only a variable percentage of fibers was found to be positive for RyR3 staining as shown in Fig. 3. A similar trend was observed in all muscles investigated including vastus lateralis, EDL, tibialis anterior, masseter, and tongue muscles (data not shown). As skeletal muscle development proceeds, the number of RyR3-containing fibers was found to decrease. Interestingly, this process was found to occur at a different rate in the different muscles analysed. Namely, while RyR3-positive fibers were found in the diaphragm and digastricus muscles of 120-day-old animals, no positive fibers were detected in the soleus muscle of animals of the same age (Fig. 3). In agreement with results obtained by Western blot analysis, RyR3-positive fibers were also detected in the masseter of adult rabbits but not in vastus, EDL, and muscles (data not shown). The percentage of RyR3-positive fibers in different rabbit muscles from 15- and 120-day-old rabbits is reported in Table 1.

RyR3 expression is restricted to slow fibers in RyR3-expressing adult skeletal muscles

The data reported in Fig. 3 indicated that the RyR3 content does not decrease gradually and simultaneously in all

Table 1
Percentage of RyR3-positive fibers in 15- and 120-day-old rabbit muscles

Muscle	D15		D120	
	Percentage	Total number of fibers	Percentage	Total number of fibers
Diaphragm	40.64	136	43.87	936
Digastricus	30.75	207	32.8	737
Soleus	27.4	269	0	100
Masseter	19.8	176	14.9	47
EDL	33.42	151	0	100
Vastus Lat.	29.8	236	0	100

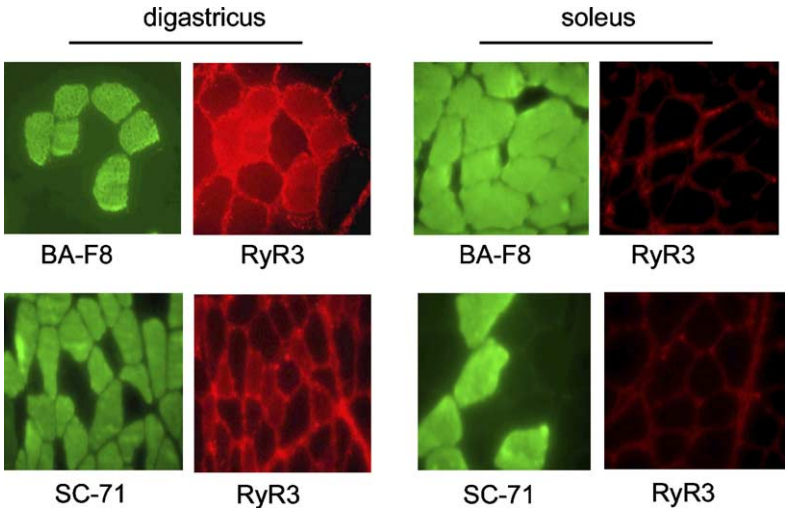

Fig. 4. Staining of paired transverse sections of adult rabbits muscles expressing RyR3 with monoclonal antibodies against slow and fast myosin isoforms in adult digastricus and soleus muscles.

muscle fibers during muscle development, suggesting that RyR3 expression in adult muscles might be preferentially restricted to specific fiber type. To verify this hypothesis, muscle sections were stained with antibodies against RyR3 and against either slow (BA-F8) or fast (SC-71) myosin isoforms. Representative sections of rabbit adult digastricus stained with anti-RyR3 polyclonal antibodies and with monoclonal antibodies against the slow or fast myosin isoforms are shown in Fig. 4. Correlation between RyR3 expression and slow myosin containing fibers could be observed in about 98% of the fibers analysed. No correlation was found between RyR3 expression and fibers expressing fast myosin isoform. It is worth noting that the correlation between RyR3 and slow myosin expressing fibers is true only for those adult muscles that express RyR3. A clear example is represented by the adult rabbit soleus muscle that, although containing a vast majority of slow myosin expressing fibers, presented no RyR3-positive fibers (Fig. 4).

Discussion

Development of skeletal muscle cells is accompanied by the regulated expression of several muscle-specific genes encoding contractile proteins and proteins involved in the regulation of Ca^{2+} homeostasis. Most muscle-specific genes start to be expressed during embryonic development, yet a significant increase in the synthesis of these proteins occurs around birth and during the neonatal period of muscle development [20]. In parallel, a developmentally regulated isoform transition of many contractile proteins can also be observed. A similar pattern may apply also to the RyR isoforms of Ca^{2+} release channels. The RyR1 isoform, which represents the main isoform of Ca^{2+} release channel in skeletal muscle, starts to be expressed shortly after muscle cells start to differentiate and its levels increase with time to attain highest levels in adult life. RyR3 channels are present in all embryonic and neonatal skeletal muscles, but not in the adult with the exception of the diaphragm muscle [11,12]. At functional level, RyR3 expression in neonatal muscle seems to contribute to the regulation of muscle contraction, as suggested by a clear reduction in the contractility observed after electrical or caffeine Ca^{2+} stimulation of muscle samples prepared from RyR3 KO mice [11]. Moreover, Ca^{2+} release studies indicated that elementary Ca^{2+} release events are dependent on the presence of RyR3 channels [21–23]. Interestingly, it has been shown that the functional expression of RyR3 channels is important to sustain the speed of Ca^{2+} release from the periphery to the center of myotubes following plasma membrane depolarization [24]. Therefore, the contribution of RyR3 channels to excitation–contraction coupling may have a specific relevance at early stages of muscle development when muscle fibers typically lack of a well-developed mature T tubule system [25].

In this study, we report that, in addition to diaphragm, the RyR3 isoform of Ca^{2+} release channels is expressed in a small number of adult skeletal muscles that include masseter, pterygoideus, digastricus, and tongue muscles. The data reported here are particularly interesting when considering the embryonic origin of RyR3-expressing muscles. While most of vertebrate muscles develop from paraxial mesoderm organized into somites, muscles of the head and neck develop either from: (1) cranial paraxial mesoderm (the occipital somitomeres); (2) mesodermal cells migrated into the branchial arches; (3) a less well-defined mesenchymal mass (septum trasversus) from which the diaphragm muscle originates [26]. Experiments with mice knockout for specific myogenic factors have clearly established that muscles that originate from the cephalic region of the embryo differ from the rest of the body musculature. Indeed, mice KO for Myf5 and Pax3 genes have shown that head muscles appear to develop independently of both Myf5 and Pax3, as mice double KO for these two genes do not present alteration of head muscles although they lack

the trunk musculature [27]. Conversely, mice double KO for MyoR and Capsulin, two genes encoding myogenic factors expressed in head and neck muscle precursor cells, show a selective ablation of facial and masticatory muscles in the presence of normal development of trunk muscles [28]. Muscles of the head and neck region differ from muscles of the trunk and limbs in the expression pattern of some muscle proteins. Indeed, head muscles are characterized by the expression of additional MHC isoforms, including MHC-α cardiac, some specific fast (extraocular and masticatory) and developmental MHC isoforms not expressed in adult muscles from other regions of the body [29–31].

In agreement with previous work on murine diaphragm [12], only a fraction of the fibers of diaphragm and of head and neck muscles of adult rabbit skeletal muscles were found to be positive for RyR3 staining. Counterstaining of paired sections with antibodies against MHC isoforms clearly established that RyR3 is expressed only in slow fibers, that is fibers positive to the BA-F8 antibody. It should be considered that the BA-F8 antibody, in addition to stain fibers containing MHC-1(also called β slow), might also recognize fibers containing MHC-α cardiac [17] which is expressed in masticatory muscles of the rabbit [32,33]. It is interesting to note that the correspondence between slow fibers and RyR3-expressing fibers occurs only in the group of muscles where RyR3 is detected. The expression of RyR3 in slow fibers of diaphragm and head muscles, but not in slow fibers of other muscles, differs from the expression of most other muscle proteins that is more strictly fiber-type dependent, irrespectively of the muscle involved. This is suggestive that there are two levels of control that regulate independently RyR3 and myosin expression in different muscles.

In conclusion, the reported results indicate that in adult animals, RyR3 Ca^{2+} release channels are present in a subset of fibers of adult muscles of cephalic origin. At the moment, while evidence is available to suggest that co-expression of RyR3 with RyR1 may be important for regulation of muscle contraction in neonatal muscle fibers, the potential role of RyR3 in adult muscles is less clear. Previous experiments performed with muscle strips prepared from adult diaphragm and EDL muscles stimulated with electrical pulses or caffeine failed to reveal any functional defect in skeletal muscles from adult RyR3 KO mice [11]. Additional studies with permeabilized muscle fibers did reveal differences between adult RyR3 KO and normal mice, and these differences were restricted to a subset of about 20% of the total population of fibers analysed [13] likely fibers expressing RyR3. Previous studies on contractile protein expression in muscle of the head and neck region [29–34] have pointed to their diversity in comparison with trunk and limb musculature: it is possible to speculate that a specific combination of RyR1 and RyR3 isoforms of Ca^{2+} release channels may be required for adapting Ca^{2+} release kinetics to the specific contractile properties of these muscles.

Acknowledgments

This work was supported by grants from Telethon (No. GGP02168), EU (HPRN-CT-2002-00331), MIUR/PRIN 2003, and MIUR/FIRB (RBNE015AX4_004) to V.S. and MIUR/FIRB (RBNE01AY9E_002) and Telethon (No. GGP04113) to C.R.

References

[1] D. Rossi, V. Sorrentino, Molecular genetics of ryanodine receptors/Ca^{2+} release channels, Cell Calcium 32 (2002) 307–319.
[2] C. Franzini-Armstrong, F. Protasi, Ryanodine receptors of striated muscles: a complex channel capable of multiple interactions, Physiol. Rev. 77 (1997) 699–729.
[3] T. Murayama, Y. Ogawa, Roles of two ryanodine receptor isoforms coexisting in skeletal muscle, Trends Cardiovasc. Med. 12 (2002) 305–311.
[4] J.L. Sutko, J.A. Airey, Ryanodine receptor Ca^{2+} release channels: does diversity in form equal diversity in function? Physiol. Rev. 76 (1996) 1027–1071.
[5] G. Giannini, E. Clementi, R. Ceci, G. Marziali, V. Sorrentino, Expression of a ryanodine receptor-Ca^{2+} channel that is regulated by TGFβ, Science 257 (1992) 91–94.
[6] G. Giannini, A. Conti, S. Mammarella, M. Scrobogna, V. Sorrentino, The ryanodine receptor/calcium channel genes are widely and differentially expressed in murine brain and peripheral tissues, J. Cell Biol. 128 (1995) 893–904.
[7] A. Conti, L. Gorza, V. Sorrentino, Differential distribution of ryanodine receptor type 3 (RyR3) gene product in mammalian skeletal muscles, Biochem. J. 316 (1996) 19–23.
[8] T. Murayama, T. Oba, E. Katayama, H. Oyamada, K. Oguchi, M. Kobayashi, K. Otsuka, Y. Ogawa, Further characterization of the type 3 ryanodine receptor (RyR3) purified from rabbit diaphragm, J. Biol. Chem. 274 (1999) 17297–17308.
[9] H. Takeshima, M. Iino, H. Takekura, M. Nishi, J. Kuno, O. Minowa, H. Takano, T. Noda, Excitation–contraction uncoupling and muscular degeneration in mice lacking functional skeletal muscle ryanodine-receptor gene, Nature 369 (1994) 556–559.
[10] J. Nakai, R.T. Dirksen, H.T. Nguyen, I.N. Pessah, K.G. Beam, P.D. Allen, Enhanced dihydropyridine receptor channel activity in the presence of ryanodine receptor, Nature 380 (1996) 72–75.
[11] F. Bertocchini, C.E. Ovitt, A. Conti, V. Barone, H.R. Scholer, R. Bottinelli, C. Reggiani, V. Sorrentino, Requirement for the ryanodine receptor type 3 for efficient contraction in neonatal skeletal muscles, EMBO J. 16 (1997) 6956–6963.
[12] B.E. Flucher, A. Conti, H. Takeshima, V. Sorrentino, Type 3 and Type 1 ryanodine receptors are colocalized in triads of the same mammalian skeletal muscle fibers, J. Cell Biol. 146 (1999) 621–629.
[13] R. Rossi, R. Bottinelli, V. Sorrentino, C. Reggiani, Response to caffeine and ryanodine receptor isoforms in mouse skeletal muscles, Am. J. Physiol. Cell Physiol. 281 (2001) C585–C594.
[14] C. DeNardi, S. Ausoni, P. Moretti, L. Gorza, M. Velleca, M. Buckingham, S. Schiaffino, Type 2X myosin heavy chain is coded by a muscle fiber type-specific and developmentally regulated gene, J. Cell Biol. 123 (1993) 823–835.
[15] S. Schiaffino, L. Gorza, S. Sartore, L. Saggin, S. Ausoni, M. Vianello, K. Gundersen, T. Lomo, Three myosin heavy chain isoforms in type 2 skeletal muscle fibres, J. Muscle Res. Cell Motil. 10 (1989) 197–205.
[16] L. Gorza, Identification of a novel type 2 fiber population in mammalian skeletal muscle by combined use of histochemical myosin ATPase and anti-myosin monoclonal antibodies, J. Histochem. Cytochem. 38 (1990) 257–265.
[17] J.J. Sciote, A. Rowlerson, Skeletal fiber types and spindle distribution in limb and jaw muscles of the adult and neonatal opossum, *Monodelphis domestica*, Anat. Rec. 251 (1998) 548–562.

[18] C.A. Lucas, J.F. Hoh, Extraocular fast myosin heavy chain expression in the levator palpebrae and retractor bulbi muscles, Invest. Ophthalmol. Vis. Sci. 38 (1997) 2817–2825.
[19] O. Delbono, G. Meissner, Sarcoplasmic reticulum Ca^{2+} release in rat slow- and fast- twitch muscles, J. Membr. Biol. 151 (1996) 123–130.
[20] S. Schiaffino, C. Reggiani, Molecular diversity of myofibrillar proteins: gene regulation and functional significance, Physiol. Rev. 76 (1996) 371–423.
[21] N. Shirokova, R. Shirokov, D. Rossi, A. Gonzalez, W.G. Kirsch, J. Garcia, V. Sorrentino, E. Rios, Spatially segregated control of Ca^{2+} release in developing skeletal muscle of mice, J. Physiol. 521 (1999) 483–495.
[22] M.W. Conklin, V. Barone, V. Sorrentino, R. Coronado, Contribution of ryanodine receptor type 3 to Ca(2+) sparks in embryonic mouse skeletal muscle, Biophys. J. 77 (1999) 1394–1403.
[23] M.W. Conklin, C.A. Ahern, P. Vallejo, V. Sorrentino, H. Takeshima, R. Coronado, Comparison of Ca (2+) sparks produced independently by two ryanodine receptor isoforms (Type 1 or Type 3), Biophys. J. 78 (2000) 1777–1785.
[24] D. Yang, Z. Pan, H. Takeshima, C. Wu, R.Y. Nagaraj, J. Ma, H. Cheng, RyR3 amplifies RyR1-mediated Ca^{2+}-induced Ca^{2+} release in neonatal mammalian skeletal muscle, J. Biol. Chem. 276 (2001) 40210–40214.
[25] A.R. Luff, H.L. Atwood, Changes in the sarcoplasmic reticulum and transverse tubular system of fast and slow skeletal muscles of the mouse during postnatal development, J. Cell Biol. 51 (1971) 369–383.
[26] D.M. Noden, Vertebrate craniofacial development: the relation between ontogenetic process and morphological outcome, Brain Behav. Evol. 38 (1991) 190–225.
[27] S. Tajbakhsh, D. Rocancourt, G. Cossu, M. Buckingham, Redefining the genetic hierarchies controlling skeletal myogenesis: Pax-3 and Myf-5 act upstream of MyoD, Cell 89 (1997) 127–138.
[28] J-R. Lu, R. Bassel-Duby, A. Hawkins, P. Chang, R. Valdez, H. Wu, L. Gan, J.M. Shelton, J.A. Richardson, E.N. Olson, Control of facial muscle development by MyoR and capsulin, Science 298 (2002) 2378–2381.
[29] A. d'Albis, C. Janmot, J.J. Bechet, Comparison of myosins from the masseter muscle of adult rat, mouse and guinea-pig. Persistence of neonatal-type isoforms in the murine muscle, Eur. J. Biochem. 156 (1986) 291–296.
[30] G.S. Butler-Browne, P.O. Eriksson, C. Laurent, L.E. Thornell, Adult human masseter muscle fibers express myosin isozymes characteristic of development, Muscle Nerve 11 (1988) 610–620.
[31] M. Monemi, P.O. Eriksson, I. Dubail, G.S. Butler-Browne, L.E. Thornell, Fetal myosin heavy chain increases in human masseter muscle during aging, FEBS Lett. 386 (1996) 87–90, [Erratum in: FEBS Lett 403 (1997) 110].
[32] J.J. Bredman, W.A. Weijs, H.A. Korfage, P. Brugman, A.F. Moorman, Myosin heavy chain expression in rabbit masseter muscle during postnatal development, J. Anat. 180 (1992) 263–274.
[33] N. Hamalainen, D. Pette, Expression of an alpha-cardiac like myosin heavy chain in diaphragm, chronically stimulated, and denervated fast-twitch muscles of rabbit, J. Muscle Res. Cell Motil. 18 (1997) 401–411.
[34] P.M. Wigmore, D.J.R. Evans, Molecular and cellular mechanisms involved in the generation of fiber diversity during myogenesis, Int. Rev. Cytol. 216 (2002) 175–232.