

CURRICULUM VITAE

Feliciano Protasi

DATI ANAGRAFICI

Data di nascita: 14 Ottobre 1966
Luogo di nascita: Foligno (PG)
Nazionalità: Italiana
Ufficio: CAST, Center for Advanced Studies and Technology
Dipartimento di Medicina e Scienze dell'Invecchiamento
Università degli Studi *Gabriele d'Annunzio*, I-66100 Chieti
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POSIZIONE ATTUALE

Dal Marzo 2011 **Professore Ordinario**, SSD BIO/09 (Fisiologia)
Università degli Studi *Gabriele d'Annunzio* (Chieti)

PARAMETRI BIBLIOMETRICI
n. pubblicazioni: 118
H index: 47
n. totale di citazioni: ~7000 (da circa ~4700 documenti)

BREVE DESCRIZIONE DELL'ATTIVITA' DI RICERCA

Il Prof. Feliciano Protasi è Professore Ordinario di Fisiologia e dirige un programma di ricerca multidisciplinare sostenuto principalmente dalla ONLUS Telethon (Italia) e dall'Istituto Nazionale di Sanità (USA).

I principali Progetti di Ricerca del suo laboratorio sono focalizzati sullo studio di:
A. malattie umane di comprovata origine genetica;
B. effetti dell'esercizio fisico sul rimodellamento delle fibre muscolari scheletriche.

Dopo la Laurea nel 1991 in Scienze Biologiche presso l'Università degli Studi di Perugia, il Dr. Protasi si è trasferito negli USA nel laboratorio della Prof.ssa Clara Franzini-Armstrong (1993-1997) presso la University of Pennsylvania (Philadelphia, PA), in cui è stato coinvolto nello studio delle differenze tra l'accoppiamento eccitazione-contrazione (EC) scheletrico e cardiaco, il meccanismo che attiva il rilascio di Ca^{2+} (e quindi la contrazione) nel muscolo. Nella seconda parte della sua esperienza all'estero (1997-2002), il Prof. Protasi si è trasferito alla Harvard Medical School (Boston, MA) nel laboratorio del Prof. Paul D. Allen in cui ha acquisito esperienza in biologia molecolare e imaging del Ca^{2+} .

Il Prof. Protasi è tornato in Italia nel 2002 come Professore Associato all'Università degli Studi Gabriele d'Annunzio per entrare a far parte del nuovo istituto CeSI (Centro Studi sull'Invecchiamento), ora CAST (Center of Advanced Studies and Technology). Stabilite le sue linee di ricerca, principalmente focalizzate nel rivelare i meccanismi pato-fisiologici alla base delle miopatie genetiche quali la suscettibilità all'ipertermia maligna (MHS) ed il colpo di calore indotto da esercizio (EHS), ora si sta occupando dello studio di un meccanismo che il muscolo utilizza per limitare la fatica (*store-operated Ca^{2+} entry*).

**INCARICHI ACCADEMICI
PRECEDENTI**

- Gen. – Giu. 2000 **Lecturer**
Harvard Medical School (Boston, MA)
- Lug. 2000 – Lug. 2002 **Instructor**
Harvard Medical School (Boston, MA)
- Mar. – Giu. 2001 **Professore a contratto**, SSD BIO/09 (Fisiologia)
Università degli Studi *Gabriele d'Annunzio* (Chieti)
- Set. 2002 - Dic. 2002 **Professore a contratto**, SSD BIO/09 (Fisiologia)
(Progetto Ministeriale: Rientro dei Cervelli).
- Dic. 2002 - Feb. 2011 **Professore Associato per chiamata diretta**, SSD BIO/09 (Fisiologia) (D.M.
MIUR prot. 1377 del 30/10/2002).

**INCARICHI DIDATTICI
ATTUALI**

- Dall'AA 2002-03 **Corso di Studi in: Scienze delle Attività Motorie e Sportive**
Titolare e Docente del Corso di Fisiologia Umana
- Dall'AA 2014-15
(fino a AA 2019-20) **Corso di Studi in: Assistenza Sanitaria**
Coordinatore del Corso Integrato di Scienze Biomediche e Fisiologiche
Docente del modulo: Fisiologia Umana
- Dall'AA 2015-16
(fino a AA 2019-20) **Corso di Laurea Magistrale in:
Scienze e Tecniche delle Attività Motorie Preventive e Adattate**
Coordinatore del Corso Integrato di Anatomia e Fisiologia
Docente del modulo: Fisiologia Umana
- Dall'AA 2019-20 **Corso di Studi in: Ostetricia**
Docente del modulo: di Fisiologia Umana
(Corso Integrato di Fisiologia e Biochimica)
- Dall'AA 2020-21 **Corso di Studi in: Medicina e Chirurgia**
Docente del modulo: Fisiologia degli Organi ed Apparati e Fisiologia Integrata
(Corso Integrato di Fisiologia 2)

**REVISORE
SCIENTIFICO**

Scientific Journals: Ageing Cell, Am J Physiol, Biophys J, Human Mut, J Cell Biol, J of Histochem and Cytochem, Pflugers Arch-European J Physiol, PNAS, Bioche J; Faseb J, Plos ONE, Human Mol Gen, Skeletal M, Cell Calcium, Oxid Med Cell Long, Nat Comm, J Mus Res Cell Motil.

Founding Agencies: Biotechnology and Biological Sciences Research Council (UK Universities); Science Foundation of Ireland; Myotubular Trust Foundation (UK); Agence Nationale de la Recherche (France).

**FINANZIAMENTI ALLA
RICERCA**

- Set. 2002 – Set. 2005
(triennale) **Finanziamento MIUR** (progetto Ministeriale *Rientro dei Cervelli*).
Titolo: *The role of Calsequestrin in skeletal EC coupling*.
Ruolo nel progetto: Principal Investigator.

- Gen. 2004 – Gen. 2006
(biennale) **Finanziamento Fondazione TELETHON ONLUS** (Progetto: GGP030289)
Progetto monocentrico coordinato da F. Protasi.
Titolo: *The role of Calsequestrin in excitation-contraction coupling and its possible contribution to skeletal muscle diseases.*
Ruolo nel progetto: Principal Investigator.
- Feb. 2007 – Feb. 2009
(biennale) **Finanziamento PRIN – MIUR** (Progetto: 2006052901_003)
Progetto Multicentrico coordinato da P. Volpe (Università di Padova)
Titolo: *Structural and functional importance of the major Ca²⁺ binding protein of the sarcoplasmic reticulum (calsequestrin) in the development and full maturation of skeletal muscle fibers.*
Ruolo nel progetto: Co-Investigator.
- Nov. 2008 – Nov. 2011
(triennale) **Finanziamento Fondazione TELETHON ONLUS** (Progetto: GGP08153)
Progetto multicentrico coordinato da F. Protasi
Titolo: *Calsequestrins in calcium homeostasis and potential role in inherited human skeletal muscle diseases.*
Ruolo nel progetto: Principal Investigator and Coordinator.
- Ago. 2010 – Giu. 2015
(quinquennale) **Finanziamento National Institute of Health - NIAMS** (Progetto: RO1 AR059646)
Progetto Multicentrico coordinato da R.T. Dirksen (Univ. of Rochester, NY).
Titolo: *Molecular Mechanism and functional role of SOCE in skeletal muscle.*
Ruolo nel progetto: Co-Investigator.
- Feb. 2011 – Gen. 2016
(quinquennale) **Finanziamento National Institute of Health - NIAMS** (Progetto: R01 AR053349)
Progetto Multicentrico coordinato da S. H. Hamilton (Baylor College, TX)
Titolo: *Basis of muscle dysfunction in Malignant Hyperthermia and Central Core Disease.*
Ruolo nel progetto: Co-Investigator.
- Ott. 2011 – Sep. 2014
(triennale) **Finanziamento Fondazione TELETHON ONLUS** (Project: GGP11141)
Progetto Multicentrico coordinato da S. Priori (Università di Pavia)
Titolo: *Mutations of cardiac calsequestrin and cardiac arrhythmias: novel insights on pathogenesis and therapy.*
Ruolo nel progetto: Co-Investigator.
- Ago. 2013 – Lug. 2016
(triennale) **Finanziamento Muscular Dystrophy Association USA** (Progetto: 275574)
Progetto Multicentrico coordinato da R.T. Dirksen (Univ. of Rochester, NY).
Titolo: *Orai1 as a Therapeutic Target for Central Core Disease.*
Ruolo nel progetto: Co-Investigator.
- Nov. 2013 – Lug. 2017
(triennale) **Finanziamento Fondazione TELETHON ONLUS** (Progetto: GGP13213)
Progetto multicentrico coordinato da F. Protasi
Titolo: *Altered calcium handling in Central Core Disease (CCD) and Malignant Hyperthermia (MH): understand molecular mechanisms and genetic background to develop innovative therapeutic interventions.*
Ruolo nel progetto: Principal Investigator and Coordinator.
- Apr. 2016 – Mar. 2021
(quinquennale) **Finanziamento National Institute of Health -NIAMS** (Progetto: RO1 AR059646-06)
Progetto Multicentrico coordinato da R.T. Dirksen (Univ. of Rochester, NY).

Titolo: *Molecular Mechanism and Functional Role of SOCE in Skeletal Muscle*.
Ruolo nel progetto: Co-Investigator.

Feb. 2017 – Gen. 2020
(triennale)

Finanziamento PRIN (Progetto: 2015ZZR4W3)
Progetto Multicentrico coordinato da V. Sorrentino (Università di Siena).
Titolo: *Novel developments in studies of Ca²⁺ entry mechanisms: relevance to skeletal muscle function and disease*.
Ruolo nel progetto: Co-Investigator.

Gen. 2020 – Dic. 2022
(triennale)

Finanziamento Fondazione TELETHON ONLUS (Progetto: GGP19231)
Progetto multicentrico coordinato da F. Protasi
Titolo: *Store-Operated Calcium Entry (SOCE): role in skeletal muscle function and disease*.
Ruolo nel progetto: Principal Investigator and Coordinator.

INVITED SPEAKER A CONGRESSI INTERNAZIONALI

Settembre 2001

30th European Muscle Conference (Pavia, PV Italy)
Titolo: *RyR/DHPR interaction in skeletal excitation-contraction coupling*.

Giugno 2003

Gordon Research Conference sull' Accoppiamento Eccitazione-Contraazione nel Muscolo (New London, NH).
Titolo: *Regions of skeletal muscle RyR and DHPR that are critical for their structural interaction*.

Settembre 2005

European Life Scientist Organization (Dresda, Germany)
Titolo: *Differences and similarities between skeletal and cardiac junctional SR: possible structural role of Calsequestrin isoforms*.

Giugno 2006

Gordon Research Conference sull' Accoppiamento Eccitazione-Contraazione nel Muscolo (New London, NH).
Titolo: *Spatial relationships between key calcium-handling molecules and organelles*.

Settembre 2006

International Symposium on Spinal Cord Motor Control (Lubiana, Slovenia)
Titolo: *Severe muscle atrophy and degeneration in spinal cord injury patients can be reversed by functional electrical stimulation (FES)*.

Marzo 2007

150th ENMC International Workshop on Core Myopathies (Naarden, The Netherlands)
Titolo: *Spatial relationships between key calcium-handling molecules and organelles in developing and adult muscle*.

Maggio 2007

2nd Basel Symposium on Skeletal Muscle (Basel, Switzerland)
Titolo. *Altered sarcotubular and mitochondrial structure and positioning in skeletal muscle fibers from animal models carrying mutations to calcium-handling proteins*.

Febbraio 2009

53rd Biophysical Society Annual Meeting (Boston, MA)
Titolo del Simposio: *Calsequestrin, triadin and more: the proteins that modulate calcium release in cardiac and skeletal muscle*. organized by Journal of Physiology, chair: Eduardo Rios and Sandor Györke.
Titolo: *Heat- and anaesthetic-induced sudden death in calsequestrin-1 knockout mice*.

- Settembre 2010 **39th European Muscle Conference** (Padova, PD Italy)
Titolo: *Calcium release units / mitochondria coupling in developing, ageing and diseased skeletal muscle.*
- Marzo 2011 **182th ENMC International Workshop on Core Myopathies** (Naarden, The Netherlands)
Titolo: *Structural association between mitochondria and sarcoplasmic reticulum.*
- Settembre 2011 **40th European Muscle Conference** (Berlin, Germany)
Titolo: *Calsequestrin-1, a new candidate gene for human muscle disorders.*
- Novembre 2012 **Société Française de Myologie** (Grenoble, France)
Titolo: *Core formation in Mouse Models of Malignant Hyperthermia and Central Core Disease.*
- Agosto 2014 **International Biophysics Congress** (Brisbane, Australia)
Titolo: *The puzzling phenotype of calsequestrin-1 knockout mice: what have we learned?*
- Ottobre 2014 **XI Meeting of the Italian Institute of Myology** (Monteriggioni, SI Italy)
Titolo: *Link between malignant hyperthermia (MH) and environmental heat stroke (EHS): just a medical hypothesis?*
- Ottobre 2014 **3rd Wiener Muskeltag** (Vienna, Austria)
Titolo: *Degeneration of chronically denervated human muscle is reversible.*
- Giugno 2015 **Gordon Research Conference on EC coupling** (Newry, ME).
Titolo: *Store operated calcium entry (SOCE) in skeletal muscle: where?*
- Dicembre 2015 **AuPS, Australian Physiological Society** (Hobart, Tasmania - Australia)
Titolo: *Exercise-dependent formation of new SR-TT junctions containing STIM1 and Orai1.*
- Febbraio 2016 **Medical School of T. Jefferson University** (Philadelphia, PA)
Titolo: *Calcium Entry Units: discovery of new intracellular junctions containing STIM1 and Orai1 in skeletal muscle.*
- Marzo 2019 **Advances in Skeletal Muscle Biology in Health and Disease** (Gainesville, FL)
Titolo: *Store-Operated Ca²⁺ Entry (SOCE) in skeletal muscle: where?*
- Ottobre 2019 **Telethon Scientific Convention** (Riva del Garda, TR Italia)
Titolo: *Store-Operated Ca²⁺ Entry (SOCE): role in Skeletal Muscle function and disease.*
- Aprile 2022 **Accademia Medica di Roma** (Roma, Italia)
Giornata a Tema: *New Frontiers in Regenerative Medicine*
Titolo: *Muscle remodeling in response to ageing, inactivity and exercise*
- ASSOCIAZIONI SCIENTIFICHE** Membro della *Biophysical Society* (dal 1998)
Membro della *Società Italiana di Fisiologia* (dal 2003)
Membro dell'*Istituto Interuniversitario di Miologia* (dal 2004)
- BIBLIOGRAFIA** Autore di 118 Pubblicazioni (10 reviews e 108 lavori originali)

BIBLIOGRAFIA COMPLETA

Reviews:

- 1 - Franzini-Armstrong, C., and F. **Protasi**. 1997. The ryanodine receptor of striated muscle: a complex channel capable of multiple interactions. *Physiol. Revs.* 77(3):699-729.
- 2 - Franzini-Armstrong, C., F. **Protasi**, and V. Ramesh. 1998. Comparative ultrastructure of calcium release units in skeletal and cardiac muscle. *Ann. NY Acad. Sci.* 853:20-31.
- 3 - **Protasi**, F. 2002. Structural interaction between RyRs and DHPRs in calcium release units of cardiac and skeletal muscle cells. In *The Structure and Function of Calcium Release Channels. Frontiers in Bioscience.* 7: d650-658.
- 4 - Fulle, S., F. **Protasi**, G. Di Tano, T. Pietrangelo, A. Beltramin, S. Boncompagni, L. Vecchiet, and G. Fanò. 2004. The contribution of reactive oxygen species to sarcopenia and muscle ageing. *Exp. Gerontol.* 39:17-24.
- 5 - Franzini-Armstrong, C., F. **Protasi**, and P. Tijssens. 2005. The assembly of Calcium Release Units in cardiac muscle. *Ann. NY Acad.* 1047:76-85.
- 6 - **Protasi**, F., C. Paolini, and M. Dainese. 2009. Calsequestrin-1: a new candidate gene for malignant hyperthermia (MH) and environmental heat stroke (EHS). *J. Physiol.* 587:3095-3100.
- 7 - **Protasi**, F., Paolini, C., M. Canato, C. Reggiani and M. Quarta. 2011. The lesson of Calsequestrin-1 ablation in vivo: much more than a buffer, after all. *J. Mus. Res. Cell Mot.* 32:257-270.
- 8 - **Protasi**, F., L. Pietrangelo, and S. Boncompagni. 2021. Calcium Entry Units (CEUs): perspectives in skeletal muscle function and disease. *J. Mus. Res. Cell Motil.* 2(2):233-249.
- 9 - **Protasi**, F., L. Pietrangelo, and S. Boncompagni. 2021. Improper remodeling of intracellular organelles deputed to Ca²⁺ handling and aerobic ATP production underlies muscle dysfunction in ageing. *Int. J. Mol. Sci.* 22(12):6195.
- 10 - Michelucci, A., C. Liang, F. **Protasi**, and R. T. Dirksen. 2021. Altered Ca²⁺ handling and oxidative stress underlie mitochondrial damage and skeletal muscle dysfunction in aging and disease. *Metabolites.* 11(7):424.

Articoli Originali:

- 1 - Sun, X-H., F. **Protasi**, M. Takahashi, H. Takeshima, D. G. Ferguson, and C. Franzini-Armstrong. 1995. Molecular architecture of membranes involved in excitation-contraction coupling of cardiac muscle. *J. Cell Biol.* 129:659-671.
- 2 - **Protasi**, F., X-H. Sun, and C. Franzini-Armstrong. 1996. Formation and maturation of calcium release apparatus in developing and adult avian myocardium. *Dev. Biol.* 173:265-278.
- 3 - Holtzer, H., T. Hijikata, Z. X. Lin, Z. Q. Zhang, S. Holtzer, F. **Protasi**, C. Franzini-Armstrong, and H. L. Sweeney. 1997. Independent assembly of 1.6 µm long bipolar MHC filaments and I-Z-I bodies. *Cell Structure and Function.* 22:83-93.
- 4 - Nakai, J., T. Ogura, F. **Protasi**, C. Franzini-Armstrong, P. D. Allen, and K. G. Beam. 1997. Functional non-equality of the cardiac and skeletal ryanodine receptors. *Proc. Natl. Acad. Sci. U.S.A.* 94:1019-1022.
- 5 - **Protasi**, F., C. Franzini-Armstrong, and B. E. Flucher. 1997. Coordinated incorporation of skeletal muscle dihydropyridine receptors and ryanodine receptors in peripheral couplings of BC₃H1 cells. *J. Cell Biol.* 137:859-870.
- 6 - Barone, V., F. Bertocchini, R. Bottinelli, F. **Protasi**, P. D. Allen, C. Franzini Armstrong, C. Reggiani, and V. Sorrentino. 1998. Contractile impairment and structural alterations of skeletal muscles from knockout mice lacking type 1 and type 3 ryanodine receptors. *FEBS letters.* 422:160-164.

- 7 - **Protasi**, F., C. Franzini-Armstrong, and P. D. Allen. 1998. Role of ryanodine receptors in the assembly of calcium release units in skeletal muscle. *J. Cell Biol.* 140:831-842.
- 8 - Franzini-Armstrong, C., F. **Protasi**, and V. Ramesh. 1999. Shapes, sizes and distributions of Ca²⁺ release units and couplons in a variety of skeletal and cardiac muscles. *Biophys J.* 77:1528-1539.
- 9 - Wang, Y., C. Fraefel, F. **Protasi**, R. A. Moore, J. D. Fessenden, I. N. Pessah, A. DiFrancesco, X. Breakefield, and P. D. Allen. 2000. HSV-1 amplicon vectors are a highly efficient gene delivery system for skeletal myoblasts and myotubes. *Am. J. Physiol. Cell Physiol.* 278:C619-6126.
- 10 - Ward, C. W., M. F. Schneider, D. Castillo, F. **Protasi**, Y. Wang, S. R. W. Chen, and P. D. Allen. 2000. Expression of ryanodine receptor 3 produces Ca²⁺ sparks in dyspedic myotubes. *J. Physiol (Lond.)*. 525:91-103.
- 11 - **Protasi**, F., H. Takekura, Y. Wang, S. R. W. Chen, G. Meissner, P. D. Allen, and C. Franzini-Armstrong. 2000. RyR₁ and RyR₃ have different roles in the assembly of calcium release units of skeletal muscle. *Biophys. J.* 79:2494-2508.
- 12 - Ward, C. W., F. **Protasi**, D. Castillo, Y. Wang, S. R. W. Chen, R. A. Moore, I. N. Pessah, P. D. Allen, and M. F. Schneider. 2001. Type 1 and type 3 ryanodine receptors generate different Ca²⁺ release event activity in both intact and permeabilized myotubes. *Biophys J.* 81:3216-3230.
- 13 - Felder, E., F. **Protasi**, R. Hirsh, C. Franzini Armstrong, and P. D. Allen. 2002. Morphology and molecular composition of sarcoplasmic reticulum surface junctions in the absence of DHPR and RyR in mouse skeletal muscle. *Biophys. J.* 82:3144-3149.
- 14 - Pietrangelo, T., M. A. Mariggio', P. Lorenzon, S. Fulle, F. **Protasi**, M. Rathbone, E. Werstiuk, and G. Fano'. 2002. Characterization of specific GTP binding sites in C2C12 mouse skeletal muscle cells. *J. Mus. Res. Cell. Motil.* 23:107-118.
- 15 - **Protasi**, F., C. Paolini, J. Nakai, K. G. Beam, C. Franzini Armstrong, and P. D. Allen. 2002. Multiple regions of RyR1 mediate functional and structural interactions with α_{15} -DHPR in skeletal muscle. *Biophys. J.* 83:3230-3244.
- 16 - Shtifman, A., C. Paolini, J. R. Lopez, P. D. Allen, and F. **Protasi**. 2004. c. *Am. J. Physiol. Cell Physiol.* 286:C73-C78.
- 17 - Lee, E. H., J. R. Lopez, J. Li, F. **Protasi**, I. N. Pessah, D. H. Kim, and P. D. Allen. 2004. Conformational coupling of DHPR and RyR1 in skeletal myotubes is influenced by long-range allostereism: evidence for a negative regulatory module. *Am. J. Physiol. Cell Physiol.* 286:C179-C189.
- 18 - **Protasi**, F., A. Shtifman, J. Julian, and P. D. Allen. 2004. All three ryanodine receptor isoforms generate rapid cooling responses in muscle cells. *Am. J. Physiol. Cell Physiol.* 286: C662-C670.
- 19 - Paolini, C., F. **Protasi**, and C. Franzini-Armstrong. 2004. The relative position of RyR feet and DHPR tetrads in skeletal muscle. *J. Mol. Biol.* 342: 145-153.
- 20 - Kern, H., S. Boncompagni, K. Rossini, W. Mayr, G. Fano', M. E. Zanin, M. Podhorska-Okolow, F. **Protasi**, and U. Carraro. 2004. Long-term denervation in humans causes degeneration of both contractile and excitation-contraction coupling apparatus that can be reversed by functional electrical stimulation (FES). A role for myofiber regeneration? *J. Neuropath. Exp. Neurol.* 63: 919-931.
- 21 - Modlin, M., C. Forstner, C. Hofer, W. Mayr, W. Richter, U. Carraro, F. **Protasi**, and H. Kern. 2005. Electrical stimulation of denervated muscles: first results of a clinical study. *Artif. Organs.* 29: 203-206.
- 22 - Fulle, S., S. Di Donna, C. Puglielli, T. Pietrangelo, F. **Protasi**, and G. Fanò. 2005. Age-dependent imbalance of the antioxidative system in human satellite cell. *Exp. Gerontol.* 40: 189-197.

- 23 - Boncompagni, S., L. d'Amelio, S. Fulle, G. Fanò, and F. **Protasi**. 2006. Progressive disorganization of the excitation-contraction coupling apparatus in ageing human skeletal muscle as revealed by electron microscopy: a possible role in the decline of muscle performance. *J. Gerontol. A Biol. Sci.* 61:995-1008.
- 24 - Ashley, Z., H. Sutherland, H. Lanmuller, M. F. Russold, E. Unger, M. Bijak, W. Mayr, S. Boncompagni, F. **Protasi**, S. Salmons, J. C. Jarvis. 2007. Atrophy, but not necrosis, in rabbit skeletal muscle denervated for periods up to one year. *Am. J. Physiol. Cell Physiol.* 292:C440-451.
- 25 - Divet, A., S. Paesante, C. Grasso, D. Cavagna, C. Tiveron, C. Paolini, F. **Protasi**, C. Huchet-Cadiou, S. Treves, and F. Zorzato F. 2007. Increased Ca²⁺ storage capacity of the skeletal muscle sarcoplasmic reticulum of transgenic mice overexpressing membrane bound calcium binding protein Junctate. *J. Cell Physiol.* 213:464-474.
- 26 - Paolini, C., M. Quarta, A. Nori, S. Boncompagni, M. Canato, P. Volpe, C. Reggiani, P. D. Allen, and F. **Protasi**. 2007. Re-organized stores and impaired calcium handling in skeletal muscle of mice lacking calsequestrin-1. *J. Physiol.* 583:767-784.
- 27 - Ashley, Z., S. Salmons, S. Boncompagni, F. **Protasi**, M.F. Russold, H. Lanmuller, W. Mayr, H. Sutherland, and J. C. Jarvis. 2007. Effects of chronic electrical stimulation on long-term denervated muscles of the rabbit hind limb. *J. Mus. Res. Cell Motil.* 28:203-217.
- 28 - Boncompagni, S., H. Kern, K. Rossini, W. Mayr, U. Carraro, and F. **Protasi**. 2007. Structural differentiation of skeletal muscle fibers in absence of innervation in humans. *Proc. Natl. Acad. Sci. USA.* 104:19339-19344.
- 29 - Kern, H., C. Hofer, M. Mödlin, W. Mayr, V. Vindigni, S. Zampieri, S. Boncompagni, F. **Protasi**, and U. Carraro. 2008. Steady state muscle atrophy in long-term paraplegics with complete upper motor neuron lesion. *Spinal Cord.* 46:293-304.
- 30 - Biral, D., H. Kern, N. Adami, S. Boncompagni, F. **Protasi**, and U. Carraro. 2008. Atrophy-resistant fibers in permanent peripheral denervation of human skeletal muscle. *Neurological Research.* 30:137-144.
- 31 - Durham, W. J., P. Aracena-Parks, C. Long, A. E. Rossi, S. A. Goonasekera, S. Boncompagni, D. L. Galvan, C. P. Gilman, N. Shirokova, F. **Protasi**, R. T. Dirksen, and S. L. Hamilton. 2008. RYR1 S-Nitrosilation underlies environmental heat stroke and sudden death in Y522S RyR1 knockin mice. *Cell.* 133:53-65.
- 32 - Rizzi, N., L. Nian, C. Napolitano, A. Nori, F. Turcato, B. Colombi, S. Biciato, D. Arcelli, A. Spedito, M. Scelsi, L. Villani, G. Esposito, S. Boncompagni, F. **Protasi**, P. Volpe, and S. G. Priori. 2008. Unexpected structural and functional consequences of the R33Q homozygous mutation in cardiac calsequestrin. A complex arrhythmogenic cascade in a knock-in mouse model. *Circulation Research.* 103:298-306.
- 33 - Dobrovolny, G., M. Augello, E. Rizzuto, S. Beccafico, C. Mammucari, S. Boncompagni, S. Belia, F. Wannenes, C. Nicoletti, Z. Del Prete, N. Rosenthal, M. Molinaro, F. **Protasi**, G. Fanò, M. Sandri, and A. Musarò. 2008. Skeletal muscle is a primary target of SOD1^{G93A}-mediated toxicity. *Cell Metabolism.* 8:425-436.
- 34 - Boncompagni, S., A. E. Rossi, M. Micaroni, G. V. Beznoussenko, R. S. Polishchuk, R. T. Dirksen, and F. **Protasi**. 2009. Mitochondria are linked to calcium stores in striated muscle by developmentally regulated tethering structures. *Mol. Biol. Cell.* 20:1058-1067.
- 35 - Dainese, M., M. Quarta, A. D. Lyfenko, C. Paolini, M. Canato, C. Reggiani, R. T. Dirksen, and F. **Protasi**. 2009. Anesthetic- and heat induced sudden death in calsequestrin-1 knockout mice. *FASEB J.* 23:1710-1720.

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