LETTER TO THE EDITOR

Fascicle length does increase in response to longitudinal resistance training and in a contraction-mode specific manner

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Dear Editor:

Morphological adaptations of skeletal muscle to resistance exercise training (RET) have been the subject of many studies: essentially, muscle hypertrophy is achieved by a structural remodelling of the contractile machinery, which can be assessed macroscopically by investigating changes in muscle architecture (i.e. fascicle length, Lf; pennation angle, PA; muscle thickness, MT) (Gans 1982; Narici 1999; Lieber and Fridén 2000, 2001; Reeves et al. 2004, 2005). A thorough understanding of muscle architecture is indeed fundamental when interpreting training-induced changes in muscle function given its key role as determinant of muscle mechanical properties (Narici et al. 2015; Lieber and Fridén 2000).

In a recent study by Fukutani and Kurihara (2015) published in SpringerPlus (2015, 4:341), the authors investigated differences in Lf between resistance trained and untrained individuals using a cross-sectional design: the main conclusion being made was that Lf was not associated with muscle hypertrophy on the basis that no significant differences in Lf were found between the groups. The authors claimed that fascicle length does not increase with resistance training.

Some fundamental considerations arise from these findings. Skeletal muscle hypertrophy in response to RET is mainly accomplished with the addition of new contractile material as a result of enhanced muscle myofibrillar protein synthesis after exercise (Glass 2003; Atherton and Smith 2012). Moreover, it is well established that the longitudinal post-natal growth of mammal muscle is associated with the increased in length and size of muscle fibres (Goldspink 1968; Williams and Goldspink 1971; Russell et al. 2000). Seminal pre-clinical studies previously showed that skeletal muscle responds to passive and intermittent stretch by adding new sarcomeres in-series (Holly et al. 1980; Goldspink 1985; Williams et al. 1988; Williams 1990), a phenomenon that occurs also in response to exercise regimes/overload, especially when including lengthening muscle actions (Goldspink 1999; Porske and Morgan 2001). Greater addition of serial sarcomeres was found in rats after downhill compared to uphill running (Lynn and Morgan 1994; Butterfield et al. 2005), reinforcing the concept of muscle longitudinal growth being intimately related to lengthening contractions. Indeed, the addition of sarcomeres in series (and thus increased Lf) appears to be one of the main "protective" mechanisms after eccentric exercise induced muscle damage (Morgan and Talbot 2002).

Further support to these observations on animal muscle can be found in numerous studies investigating architectural responses to RET, directly in humans. Interestingly, Fukutani and Kurihara stated it as controversial as to whether Lf increases after RET: however the number of reports showing no increases in Lf in response to exercise is limited (Blazevich et al. 2007b; Erskine et al. 2010; Ema et al. 2013) compared to those that demonstrated an increase in Lf after either conventional resistance, isokinetic, isoinertial or even marathon training (Morgan and Porske 2004; Seynnes et al. 2007; Blazevich et al. 2007a; Potier et al. 2009; Reeves et al. 2009; Baroni et al. 2013; Franchi et al. 2014, 2015; Sharifnezhad et al. 2014; McMahon et al. 2014; Murach et al. 2015). But, most importantly, it was recently reported by our group that, in both young and older populations, architectural changes, such as increases in Lf, are somewhat contraction-specific (Reeves et al. 2009; Franchi et al. 2014, 2015). That is, concentric loading promotes increases in PA, reflecting preferential addition of sarcomeres in parallel, whereas eccentric training favours the increase of...
Lf through the addition of sarcomeres in series. It is our opinion that these investigations should have been cited in Fukutani and Kurihara’s manuscript. Furthermore, considering the substantial number of longitudinal studies that have shown significant changes in Lf and muscle architecture after RET, the adoption of such a cross-sectional study design calls into question the validity of these conclusions. Moreover, the investigation was performed on recreationally active volunteers (the untrained group, with “no experience in regular RET”) compared to a group of “resistance exercise trained” participants, either bodybuilders or rugby players (i.e. the number of bodybuilders/rugby players was not specified). Taking into account the aforementioned considerations on the contraction-specificity of architectural responses, the individual history of resistance training in both groups should have been accounted for. Kawakami and colleagues (1993) previously reported that PA and MT are greater in bodybuilders compared to untrained and moderately trained subjects (Lf was not investigated), but Abe et al. (2000, 2001), showed that Lf is greater in elite male 100 m-sprinters compared to elite long-distance runners and to non-sprinters. Rather than being innate factors, as Fukutani and Kurihara argue, architectural adaptations such as increases in Lf are indeed detectable longitudinally and are training/traction-specific (Blazevich et al. 2003; Franchi et al. 2014, 2015). In addition, Lf was measured as a straight line in Fukutani and Kurihara’s study: while this might not represent a problem in the untrained group, in hypertrophied muscle, instead, fas-cicles show a significantly greater curvature, which partially explains the increased pennation occurring with hypertrophy (clearly visible in bodybuilders muscle) (Kawakami et al. 1993). Since the fascicle curvature was neglected by the methodological approach used to measure Lf, the true Lf values could have been underestimated in the resistance-trained group. Therefore, Lf may have gone undetected as a result of the simplicity of the morphometric analyses implemented. Thus, the chances are that Fukutani and Kurihara’s results were biased by the non-longitudinal study design and by the possible under-estimation of Lf due to specific methodological approach. We agree that in some cases “muscle hypertrophy is not necessarily accompanied with increase in Lf” (Fukutani and Kurihara 2015), but these cases can only be truly determined by tightly controlled longitudinal studies.

We are of the opinion that the nature of fascicle length (Lf) increase is highly dependent on which type of contraction and mechanical stimulus is predominant in specific RET programmes: thus, data on muscle architecture features should be cautiously interpreted, as crucial in the understanding of muscle structural remodelling and its functional characteristics.

**Abbreviations**

RET: resistance-exercise training; Lf: fascicle length; PA: pennation angle; MT: muscle thickness.

**Authors’ contributions**

MVF: conception of the manuscript; MVF, PJA, CNM and MVN: drafting of the manuscript. All authors read and approved the final manuscript.

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**Competing interests**

The authors declare that they have no competing interests.

**Funding information**

We acknowledge the Biotecnology and Biological Research Council (BBSRC) UK. Some of the referenced data were obtained from a BBSRC UK funded project (code: BB/K01914/1).

**Received: 1 September 2015 Accepted: 20 November 2015**

Published online: 28 January 2016

**References**

Abe T, Kumagai K, Brechue WF (2000) Fascicle length of leg muscles is greater in sprinters than distance runners. Med Sci Sports Exerc 32:1125–1129

Abe T, Fukashiro S, Harada Y, Kawamoto K (2001) Relationship between sprint performance and muscle fascicle length in female sprinters. J Physiol Anthropol Appl Human Sci 20:141–147

Atherton PJ, Smith K (2012) Muscle protein synthesis in response to nutrition and exercise. J Physiol 590:1049–1057. doi:10.1113/jphysiol.2011.225003

Baron BM, Geremia JM, Rodrigues R et al (2013) Muscle architecture adaptations to knee extensor eccentric training: rectus femoris vs vastus lateralis. Muscle Nerve. doi:10.1002/mus.23785

Blazevich AJ, Gill ND, Bronks R, Newton RU (2003) Training-specific muscle architecture adaptation after 5-week training in athletes. Med Sci Sports Exerc 35:2013–2022. doi:10.1249/01.MSS.0000090902.83611.20

Blazevich AJ, Cunnan D, Coleman DR, Horne S (2007a) Influence of concentric and eccentric resistance training on architectural adaptation in human quadriceps muscles. J Appl Physiol 103:1565–1575. doi:10.1152/japplphysiol.00578.2007

Blazevich AJ, Gill ND, Dears N, Zhou S (2007b) Lack of human muscle architec-tural adaptation after short-term strength training. Muscle Nerve 35:78–86. doi:10.1002/mus.20666

Butterfield TA, Leonard TR, Herzog W (2005) Differential serial sarcomere number adaptations in knee extensor muscles of rats is contraction type dependent. J Appl Physiol. doi:10.1152/japplphysiol.00481.2005

Ema R, Wakahara T, Miyamoto N et al (2013) In homogogeneous architectural changes of the quadriceps femoris induced by resistance training. Eur J Appl Physiol 113:2691–2703. doi:10.1007/s00421-013-2700-1

Erskine RM, Jones DA, Williams AG et al (2010) Inter-individual variability in the adaptation of human muscle specific tension to progressive resistance training. Eur J Appl Physiol 110:1117–1125. doi:10.1007/s00421-010-1601-9

Franchi MV, Atherton PJ, Reeves ND et al (2014) Architectural, functional and molecular responses to concentric and eccentric loading in human skeletal muscle. Acta Physiol 210:642–654. doi:10.1111/apha.12225

Franchi MV, Wilkinson DJ, Quinlan JI et al (2015) Early structural remodelling and deuterium oxide-derived protein metabolic responses to eccentric and concentric loading in human skeletal muscle. Physiol Rep. doi:10.14814/phy2.12593

Fukutani A, Kurihara T (2015) Comparison of the muscle fascicle length between resistance-trained and untrained individuals: cross-sectional observation. Springerplus 4:341. doi:10.1186/s40064-015-1133-1

Gans C (1982) Fiber architecture and muscle function. Exerc Sport Sci Rev 10:160–207
Glass DJ (2003) Molecular mechanisms modulating muscle mass. Trends Mol Med 9:344–350. doi:10.1016/S1471-4914(03)00138-2
Goldspink G (1968) Sarcomere length during post-natal growth of mammalian muscle fibres. J Cell Sci 3:539–548
Goldspink G (1985) Malleability of the motor system: a comparative approach. J Exp Biol 115:375–391
Goldspink G (1999) Changes in muscle mass and phenotype and the expression of autocrine and systemic growth factors by muscle in response to stretch and overload. J Anat 194(3):323–334
Holly RG, Barnett JG, Ashmore CR et al (1980) Stretch-induced growth in chicken wing muscles: a new model of stretch hypertrophy. Am J Physiol 238:C62–C71
Kawakami Y, Abe T, Fukunaga T (1993) Muscle-fiber pennation angles are greater in hypertrophied than in normal muscles. J Appl Physiol 74:2740–2744
Lieber RL, Fridén J (2000) Functional and clinical significance of skeletal muscle architecture. Muscle Nerve 23(11):1647–1666. doi:10.1002/1097-4598(200011)23:11<1647:AID-MUS1>3.0.CO;2-M
Lieber RL, Fridén J (2001) Clinical significance of skeletal muscle architecture. Clin Orthop Relat Res 383:140–151
Lynn R, Morgan DL (1994) Decline running produces more sarcomeres in rat vastus intermedius muscle fibers than does incline running. J Appl Physiol 77(3):1439–1444
McMahon GE, Morse CI, Burden A, Winwood K, Onambélé GL (2014) Impact of range of motion during ecologically valid resistance training protocols on muscle size, subcutaneous fat, and strength. J Strength Cond Res 28(1):245–255
Morgan DL, Proske U (2004) Popping sarcomere hypothesis explains stretch-induced muscle damage. Clin Exp Pharmacol Physiol 31:541–545. doi:10.1111/j.1440-1681.2004.04029.x
Morgan DL, Talbot JA (2002) The addition of sarcomeres in series is the main protective mechanism following eccentric exercise. J Mech Med Biol 02:421–431. doi:10.1142/S0219519402000423
Murach K, Grieve C, Luden ND (2015) Skeletal muscle architectural adaptations to marathon run training. Appl Physiol Nutr Metab 40:99–102. doi:10.1139/apnm-2014-0287
Narici M (1999) Human skeletal muscle architecture studied in vivo by non-invasive imaging techniques: functional significance and applications. J Electromyogr Kinesiol. doi:10.1016/S1050-6411(98)00041-8
Narici MV, Franchi MV, Maganaris CN (2015) Muscle structural assembly and functional consequences. J Exp Biol (Epub ahead of print)
Potier TG, Alexander CM, Seynnes OR (2009) Effects of eccentric strength training on biceps femoris muscle architecture and knee joint range of movement. Eur J Appl Physiol 105:939–944. doi:10.1007/s00421-008-0980-7
Proske U, Morgan DL (2001) Muscle damage from eccentric exercise: mechanism, mechanical signs, adaptation and clinical applications. J Physiol 2:333–345
Reeves ND, Narici MV, Maganaris CN (2004) In vivo human muscle structure and function: adaptations to resistance training in older humans. Exp Physiol 89:675–689
Reeves ND, Maganaris CN, Narici MV (2005) Plasticity of dynamic muscle performance with strength training in elderly humans. Muscle Nerve 31:355–364. doi:10.1002/mus.20275
Reeves ND, Maganaris CN, Longo S, Narici MV (2009) Differential adaptations to eccentric versus conventional resistance training in older humans. Exp Physiol 94:825–833. doi:10.1113/expphysiol.2009.046599
Russell B, Matlagh D, Ashley WW (2000) Form follows function: how muscle shape is regulated by work. J Appl Physiol 88:1127–1132
Seynnes OR, De Boer M, Narici MV (2007) Early skeletal muscle hypertrophy and architectural changes in response to high-intensity resistance training. J Appl Physiol 102(1):368–373. doi:10.1152/japplphysiol.00789.2006
Sharifnejhad A, Maraliger R, An rampatzis A (2014) Effects of load magnitude, muscle length and velocity during eccentric chronic loading on the longitudinal growth of vastus lateralis muscle. J Exp Biol. doi:10.1242/jeb.100370
Williams PE (1990) Use of intermittent stretch in the prevention of serial sarcomere loss in immobilised muscle. Ann Rheum Dis. doi:10.1136/ard.49.5.316
Williams PE, Goldspink G (1971) Longitudinal growth of striated muscle fibres. J Cell Sci 9:751–767
Williams PE, Catenese T, Lucey EG, Goldspink G (1988) The importance of stretch and contractile activity in the prevention of connective tissue accumulation in muscle. J Anat 158:109–114