Original article

Agenesis of palmaris longus muscle: is this a phenotype of variable expressivity?

Morais, MA.[1], Santos, WG.[2] and Malysz, T.[1]*

[1]Laboratório de Anatomia Humana e Comparada, Universidade Federal de Goiás – UFG, Rod BR 364 Km 192, Setor Parque Industrial, 3800, CP 03, CEP 75800-000, Jataí, GO, Brasil
[2]Laboratório de Genética, Universidade Federal de Goiás – UFG, Rod. BR 364 Km 192, Setor Parque Industrial, 3800, CP 03, CEP 75800-000, Jataí, GO, Brasil
*E-mail: taismalysz@yahoo.com.br

Abstract

Introduction: the aim of this study was to investigate the putative hereditary pattern related to the presence/absence of the palmaris longus muscle (PLM) in humans. **Materials and Methods:** we analyzed 99 individuals (56 women and 43 men) from 25 families living in the city of Jataí, southwestern of Goiás State, Brazil, who agreed to participate in the study by signing an approved consent form according to the University Research Ethical Committee. The tests to detect the presence/absence of PLM were performed in all subjects in both forearms by clinical examination. A careful analysis of all families phenotypes studied was performed. **Results:** Bilateral presence of PLM was detected in the majority of the population studied (75.76%). The unilateral absence of PLM was observed in 14.14% of subjects and bilateral absence in 10.1%. In 14 families both parents and 25 children had bilateral PLM and one child had unilateral absence of PLM. Parents Families (n=9) where one parent showed bilateral presence and the other unilateral/bilateral absence were observed children with both bilateral presences, unilateral or bilateral absence of PLM. Cases where both parents showed unilateral absence of PLM with children showing bilateral absence or presence of PLM were also observed. **Conclusion:** The presence of PLM appears to be dominant over its absence. It is suggested that the genotype for the absence of muscle is recessive while for the presence is dominant but with variable expressivity for the phenotype of unilateral or bilateral presence of the muscle.

Keywords: agenesis, phenotype, genotype, palmaris longus muscle.

1 Introduction

Anatomically, the palmaris longus muscle (PLM) is described as a small fusiform muscle located superficially in the anterior forearm, between the flexor carpi radialis muscle and the flexor carpi ulnaris muscle. It arises from the medial epicondyle of the humerus by the common flexor tendon, from the intermuscular septa between it and the adjacent muscles, and from the antebrachial fascia. It is inserted by a long tendon into the distal half of the flexor retinaculum and lower part of the palmar aponeurosis (MOORE and DALLEY, 2007). Functionally, the palmaris longus muscle seems to contribute little to the wrist flexion (MOORE and DALLEY, 2007). Furthermore, it acts in tension of the palmar aponeurosis and at the increased strength of thumb abduction (FAHRER, 1973; GANGATA, NDOU and LOUW, 2010).

The PLM is known as one of the most variable muscles of the human body (KOO and ROBERTS, 1997; YILDIZ, SNER and AYNACI, 2000). The variations include changes in location and shape of their fixations and their muscular bundle, presence of fascicles accessories and complete agenesis (KOO and ROBERTS, 1997; KAWASHIMA, KIKUSHIMA, YOKOTA et al., 2002).

High prevalence (24%) of the PLM agenesis was reported in North American Caucasians (TROHA, BAIBAK and KELLEHER, 1990). A recent study described the Turkish population with a prevalence of 26.6% of cases of agenesis (KOSE, ADANIR, CIRPAR et al., 2009). Studies among the Asian population showed that the incidence is 3.4% in Japan (ADACHI, 1909), 4.6% in the Chinese (SEBASTIN, PUHAINDRAN, LIM et al., 2005) and 20.2% in the Indians (AGARWAL, 2010). In Brazil, our recent study shows a prevalence of 12.2% and 14.3% of bilateral and unilateral agenesis of palmaris longus, respectively (MORAIS, GOMES, HELRIGLE et al., 2012). Furthermore its absence is most common in women and on the left side (GOSCICKA, STEPIEN and GOSCICKA, 1981), however, studies on this issue are still controversial (SEBASTIN, PUHAINDRAN, LIM et al., 2005; GARCIA, MARQUES, SANTOS et al., 2005).

Although no major functional relevance to PLM has been described, at least to the clinical point of view, there is a growing interest in the presence of PLM tendon in the forearm, because it has been often used for interventions and reconstructive plastic surgery of the hand (SEBASTIN, PUHAINDRAN, LIM et al., 2005). Additionally, the tendon has been used in a wide variety of procedures including lip augmentation (DAVIDSON, 1995), correction of ptosis (NAUGLE and FAUST, 1999) and restoration of facial paralysis (ATIYEH, HASHIM, HAMDAN et al., 1998).

The curiosity about the causative or associated factors related to the determination of agenesis of PLM has aroused the interest of many researchers. In an attempt to clarify the occurrence of such variation, some researchers believed that there could be correlated with the presence of other

associated anatomical variations that include the presence of anomalous superficial of the palmar arch (O'SULLIVAN and MITCHELL, 2002) and the absence of the plantaris muscle (VANDERHOOFT, 1996). However, no variation associated has been confirmed.

Some authors have suggested a possible association of agenesis of the PLM with a pattern of inheritance (THOMPSON, MCBATTS and DANFORTH, 1921; DANFORTH, 1924; MUNTONI, BROWN, SEWRY et al., 2002). However, it is still unclear what type of inheritance is involved or what are the supposed genes involved in this human muscle variation.

Given the scarcity of research in the area, this study aimed to investigate the possible inheritance pattern involved in the transmission of the presence/absence of the palmaris longus muscle in a group of families residing in the southwestern part of the Goiás State, Brazil.

2 Materials and Methods

This research was conducted in the city of Jataí-Goiás State, Brazil and was, previously approved by the Ethics Committee of Universidade Federal de Goiás (protocol 167/2010). The sample comprised 99 individuals (56 women and 43 men) from 25 families of Jataí, who agreed to participate by signing an appropriate consent form. Subjects with a history of injury, disease or abnormality of at least one upper limb, which made difficult or precluded the achievement of flexion of the fingers and wrist, were excluded from the study.

The test to detect the presence/absence of PLM was performed in parents and children from 25 families in both forearms by clinical examination. The subject was initially submitted to the standard test for identification of PLM tendon. For this, the individual subject was instructed to realize the opposition the first and fifth finger and sequentially perform wrist flexion (SCHAEFFER, 1909). If the muscle had not been visualized or palpable through this test, another test was conducted which consisted of the additional flexion of the fingers II to V, followed by wrist flexion and realization of opponency and flexion of the thumb on the other fingers (THOMPSON, MCBATTS and DANFORTH, 1921).

The data from all individuals were recorded and families pedigree were created. Descriptive statistics was used to analyze the data and a careful analysis of all families phenotypes studied was performed.

3 Results

The majority of individuals who participated in the study showed bilateral presence of PLM (75 individuals, 75.76%). The unilateral absence of the muscle was observed in 14 individuals (14.14%) while bilateral absence was observed in 10 individuals (10.1%).

The most frequent observation among the 25 families analyzed was the occurrence of both parents showing bilateral presence of PLM and having children with bilateral PLM as well (Table I). In 14 families both parents and 25 children had bilateral PLM and one child had unilateral absence of PLM (Figures 1A and 1C). Also, in families where one of the parents showed bilateral presence and the other showed unilateral/bilateral absence were observed children with bilateral, unilateral or bilateral absence of PLM (Figure 1A-B). Additionally, we found that parents with unilateral absence of PLM had children with bilateral absence or presence of the same (Figure 1A, Table 1).

4 Discussion

The abnormal development of skeletal muscle, observed in some human clinical conditions, possibly involves changes in genes supposedly related to muscle growth. Some of these conditions occur sporadically while others are hereditary or part of a dysmorphic syndrome (MUNTONI, BROWN, SEWRY et al., 2002). The Dysmorphic syndromes, characterized by absence or hypoplasia of skeletal muscles, include the Holt-Oran syndrome, Fyns syndrome, Velocardiofacial syndrome, and possibly agenesis of palmaris longus muscle (MUNTONI, BROWN, SEWRY et al., 2002).

It is known that during the embryonic development, the somitic cells of the mesoderm depend of the positive and negative signals from surrounding tissues necessary to activate the genes capable to transform them in muscle cells (DAL PAI-SILVA and CARVALHO, 2007). These cells must be precommitted to the myogenic lineage through the transcription factor Pax3 (GOULDING, LUMSDEN and PAQUETTE, 1994).

The genes responsible for this transformation are members of the family of transcription factors that share a basic domain helix-loop-helix (bHLH) required for binding with DNA and dimerization with transcriptional factors of the protein E family. This family is part of the MyoD, Myogenin, Myf5 and MRF4, called myogenic regulatory factors (MRFs) (MURRE, McCAW, VAESSIN et al., 1989;

Table 1. Inheritance pattern of palmaris longus muscle.

PARENTS		Number of subjects	Number of families	CHILDREN							
Parental Matings				Boys				Girls			
Father	Mother			PP	PA	AP	AA	PP	PA	AP	AA
PP	PP	54	14	10	0	0	0	15	0	1	0
PP	PA	11	3	1	0	0	0	3	0	1	0
PP	AA	5	1	1	0	0	0	0	0	0	2
AP	PP	12	3	0	1	0	0	3	1	0	1
AA	PP	12	3	0	1	0	0	3	1	0	1
PA	PA	5	1	1	0	0	1	0	0	0	1

P: presence; A: absence of palmaris longus muscle. The first position of the letter indicates the right forearm and the second the left forearm.

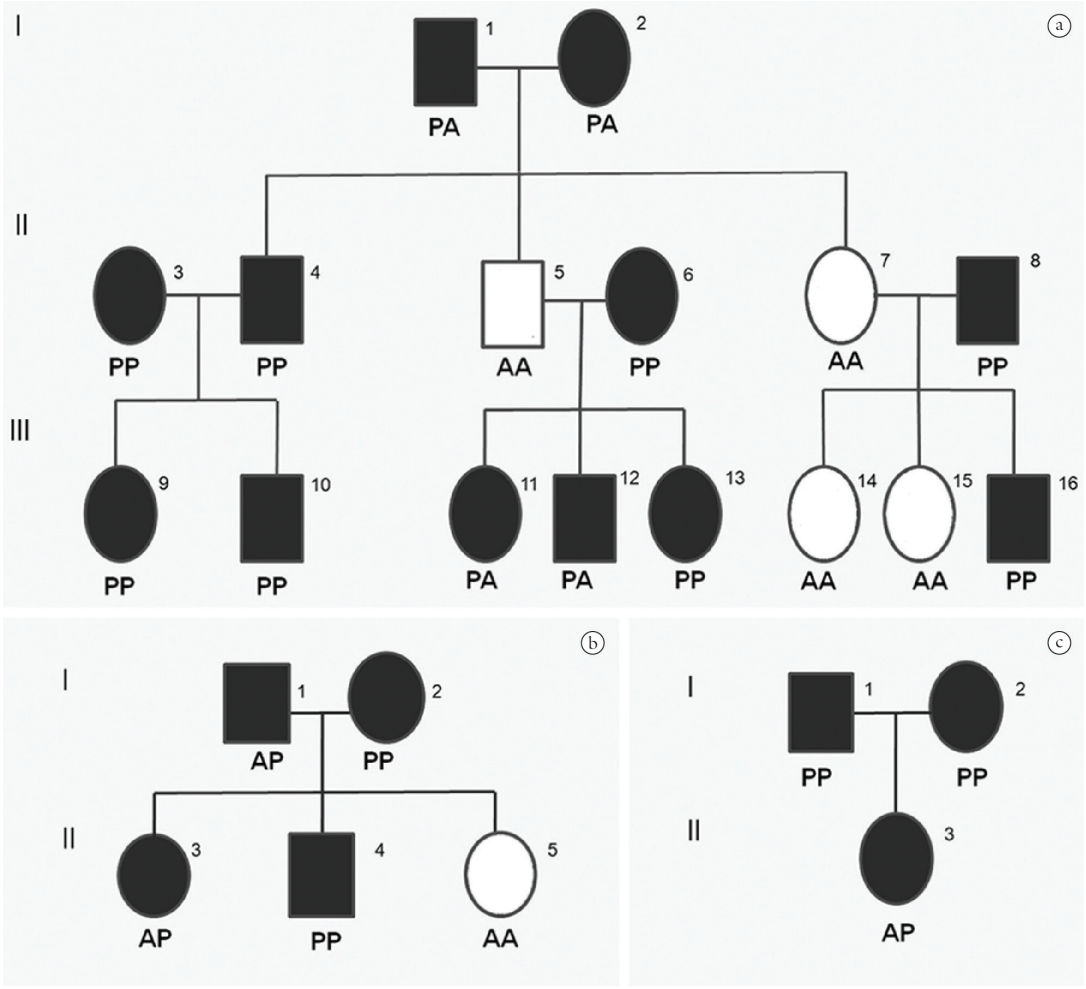

Figure 1. Family Pedigree of the families analyzed (A, B, C) depicting the pattern of inheritance of palmaris longus muscle. Squares: men; Circles: women. Filled symbols indicate presence unilateral/bilateral of PLM, open symbols absence bilateral of PLM. Marriage, children and subjects parent degree are indicated with the standard genetic symbols. P represents presence and A absence of palmaris longus muscle. The first position of the letter indicates the right forearm and the second the left forearm, e.g. PA indicates presence of the muscle in the right forearm and absence in the left.

TAJBAKHSH, ROCANCOURT, COSSU et al., 1997). The myoblasts leaving the cell cycle and expressing MyoD and Myf5, become differentiated myocytes and start the expression of MRFs MRF4 and myogenin, which regulate the differentiation of these cells in muscle fibers (MEGENEY and RUDNICKI, 1995).

Like the MRFs, the MEF2 (Myocyte Enhancer Factor-2) family of transcription factors is also involved in the activation of muscle genes-specific (NAYA and OLSON, 1999). Studies show an interdependent action between MRFs and MEF2 families in controlling the differentiation of skeletal muscle (NAIDU, LUDOLPH, TO et al., 1995).

Different authors agree on the influence of hereditary factors on the absence of the muscle (THOMPSON, MCBATTS and DANFORTH, 1921; DANFORTH, 1924; WEHBE, 1992; GARCIA, MARQUES, SANTOS et al., 2005). The first author who suggested the suppression of PLM as dependent of hereditary factors was Thompson, Mcbatts and Danforth (1921), by analysis of the phenotype in 102 families. However, how this suppression occurs by a genetic mechanism is still unclear (WEHBE, 1992).

Few studies have addressed the hereditary component of the PLM agenesis. The most frequent report is related to the presence of PLM in most individuals of the same family. In our study, 13 (52%) out of the 25 families surveyed, showed both parents and 25 children presenting the PLM bilaterally. According to Thompson, Mcbatts and Danforth (1921), of the 102 families, 68 (66.6%) had the same phenotype. On the other hand, from the 25 families studied by Garcia, Marques, Santos et al. (2005), 21 of them (84%), showed parents and the children with bilateral presence of the muscle.

In the present study, we found that parents with bilateral presence of PLM can generate children with unilateral absence. In families where one of the parents showed bilateral presence and the other showed unilateral/bilateral absence, we could detect children with both bilateral presence or unilateral/bilateral absence of PLM. Additionally, we found in our population studied parents with unilateral absence of PLM had who had children with bilateral absence or presence of the same, but not with unilateral absence. These results also have been described by Thompson, Mcbatts

and Danforth (1921), in family pedigree analysis of the 33 families that have generated 67 children.

We believe that the gene or the genes that determine the characteristics of presence and absence of PLM may not be considered exclusively dominant or recessive given the fact that children may have phenotypes different from those observed in their parents. These different phenotypes do not follow those expected for a feature purely dominant or recessive according to Mendelian inheritance patterns. A similar analysis conducted by Thompson, Mcbatts and Danforth (1921) led them to infer about the existence of many unknown factors that influence the genes that determine the agenesis of palmaris longus muscle. Danforth (1924), analyzing the data from Thompson also suggested the existence of factors that influence the presence or absence of PLM, which he called the modifying factors. Some of these would be considered relatively powerful while others would be weak in interfering with the development of the bilateral or unilateral agenesis of the muscle.

It is known that is not always possible to establish a direct relationship between genotype and phenotype due to variation in expression of a gene. The variable expressivity has been described in humans, animals and plants whose alleles of many genes can trigger or cause varying severity in the phenotype. The effects are attributed to interactions with other genes, with environmental factors or to the influence of epigenetics factors on gene expression (GRIFFITHS, WESSLER, LEWONTIN et al., 2008; LALUCQUE and SILAR, 2004).

In Neurofibromatosis type I, a disease showing variable expressivity, the parent with no clinical detection or manifestation of the disease is capable of transmitting the mutant allele to a descendant that show a serious condition as result of the expression of the mutation. This type of gene interaction can also be exemplified by sickle cell disease, Pfeiffer syndrome, Crouzon syndrome and Jackson-Weiss (REGATEIRO, 2007).

Osteogenesis imperfecta, with autossomal dominant transmission, also illustrates the concept o variable expressivity. In genealogies ascertained from patients with this heredopathia the individuals demonstrate bone fragility, blue sclerae and deafness consequent to otosclerosis, along with consanguineous relatives who exhibit all these signs or just one. Thus, it is possible to say that the disease can be manifested by one, two or three of the main clinical signs of the syndrome in individuals belonging to the same genealogy, and that these signs can have variable intensity (BEIGUELMAN, 2008). Similarly, other medical conditions have variable phenotypic expression, including Waardenburg syndrome (BARZOTTO and FOLADOR, 2004), Marfan syndrome (PEREIRA, D'ALESSIO, RAMIREZ et al., 1993) and the little finger Camptodactyly (BEIGUELMAN, 2008).

The fact that most of the subjects in the population studied in this work presented the PLM at least in one of the forearms suggests that the presence of the muscle is dominant over its absence. However, according to the phenotype analysis of family members in the studied group, we found that the unilateral or bilateral presence of the muscle cannot be considered neither homozygous dominant (*DD*) nor heterozygous (*Dd*) as would be expected for an inheritance purely dominant. Thus, we suggest that the presence of PLM is a case of autossomal dominant inheritance with variable expressivity. Therefore, the genotype for the presence of PLM could be expressed for instance as *D-*, considering that the *D* allele can express variable phenotypes such as unilateral or bilateral presence of the muscle and the genotype for bilateral absence of the muscle could be referred as *dd*.

We therefore believe that the variable expressivity may explain all the variable phenotypes showed by parents and children in different families analyzed in this study, and also in the studies conducted by Thompson, Mcbatts and Danforth (1921) and Garcia, Marques, Santos et al. (2005). Although the gene or genes associated with the determination of unilateral or bilateral agenesis of the palmaris longus muscle has not yet been described, we believe that the changes are associated with the myogenic regulatory transcription factors and further studies need to be conducted to better understand the molecular mechanism of this intriguing genetic issue.

5 Conclusion

From the analysis of phenotypes of the families studied we found that the presence of PLM appears to be dominant over its absence. It is suggested that the genotype for the bilateral absence of muscle could be referred as recessive and for the presence of muscle as dominant with variable expressivity for the phenotype which could show as unilateral or bilateral presence of the muscle in the forearm.

However, to confirm the real genotypes, further studies should be conducted aimed at identifying genes responsible and/or involved with agenesis of the palmaris longus muscle.

References

ADACHI, B. Beitrage zur Anatomie der Japaner. XII. Die Statistic der Muskelvarietäten zweite Mitteilung. *Zeitschrift Für Morphologie und Anthropologie*, 1909, vol. 12, n. 2, p. 261-312.

AGARWAL, P. Absence of the palmaris longus tendon in Indian population. *Indian Journal of Orthopaedics*, 2010, vol. 44, n. 2, p. 212-215. PMid:20419011 PMCid:PMC2856399. http://dx.doi.org/10.4103/0019-5413.61863

ATIYEH, BA., HASHIM, HA., HAMDAN, AM., KAYLE, DL. and MUSHARAFIEH, RS. Lower reconstruction and restoration of oral competence with dynamic palmaris longus vascularised sling. *Archives of Otolaryngology-Head & Neck Surgery*, 1998, vol. 124, n. 12, p. 1390-2. PMid:9865766. http://dx.doi.org/10.1001/archotol.124.12.1388

BARZOTTO, JDV. and FOLADOR, MF. Síndrome de Waardenburg: características audiológicas. *Revista CEFAC*, 2004, vol. 6, n. 3, p. 306-311.

BEIGUELMAN, B. *A interpretação genética da variabilidade humana*. Ribeirão Preto: Sociedade Brasileira de Genética, 2008. 152 p.

DAL PAI-SILVA, M. and CARVALHO, RF. Mecanismos celulares e moleculares que controlam o desenvolvimento e o crescimento muscular. *Revista Brasileira de Zootecnia*, 2007, vol. 36, p. 21-31. http://dx.doi.org/10.1590/S1516-35982007001000003

DANFORTH, CH. The hereditary of unilateral variations in man. *Genetics*, 1924, vol. 9, n. 3, p. 199-211.

DAVIDSON, BA. Lip augmentation using the Palmaris longus tendon. *Plastic Reconstrutive Surgery*, 1995, vol. 95, n. 2, p. 1108-1110. PMid:7732124.

FAHRER, M. Proceedings: The role of the palmaris longus muscle in the abduction of the thumb. *Journal of Anatomy*, 1973, vol. 116, p. 476. PMid:4791423.

GANGATA, H., NDOU, R. and LOUW, G. The Contribution of the Palmaris Longus Muscle to the Strength of Thumb Abduction. *Clinical Anatomy*, 2010, vol. 23, n. 4, p. 431-436. PMid:20235177. http://dx.doi.org/10.1002/ca.20960

GARCIA, LB., MARQUES, CN., SANTOS, CM. and BERTOLINI, SMMG. *Estudo da prevalência do músculo palmar longo em humanos.* Iniciação Científica CESUMAR, 2005. vol. 7, p. 19-24.

GOSCICKA, D., STEPIEN, J. and GOSCICKA, J. Long palmar muscle in human fetuses. *Gegenbaurs Morphologisches Jahrbuch*, 1981, vol. 127, n. 2, p. 292-9. PMid:7250662.

GOULDING, M., LUMSDEN, A. and PAQUETTE, A.J. Regulation of Pax-3 expression in the dermomyotome and its role in muscle development. *Development*, 1994, vol. 120, n. 4, p. 957-971. PMid:7600971.

GRIFFITHS, JF., WESSLER, R., LEWONTIN, C. and CARROL, B. *Introdução à genética*. 9. ed. Rio de Janeiro: Guanabara Koogan, 2008. 726 p.

KAWASHIMA, T., KIKUSHIMA, S., YOKOTA, E., OHKUBO, F., YAMANA, Y., SATO, F. and SASAHI, H. A case of an accessory palmaris longus muscle and a duplicate palmaris longus muscle with special reference to their nerve supply-morphologic significance of a common innervation trunk. *Okajimas Folia Anatomica Japonica*, 2002, vol. 79, n. 2-3, p. 75-81. PMid:12425381. http://dx.doi.org/10.2535/ofaj.79.75

KOO, CC. and ROBERTS, AH. The palmaris longus tendon. Another variation in its anatomy. *Journal of Hand Surgery*, 1997, vol. 22, n. 1, p. 138-9. PMid:9018627.

KOSE, O., ADANIR, O., CIRPAR, M., KURKLU, M. and KOMURCU, M. The prevalence of absence of the palmaris longus: a study in Turkish population. *Archives of Orthopaedic and Trauma Surgery*, 2009, vol. 129, n. 5, p. 609-11. PMid:18418616. http://dx.doi.org/10.1007/s00402-008-0631-9

LALUCQUE, H. and SILAR, P. Incomplete Penetrance and Variable Expressivity of a Growth Defect as a Consequence of Knocking Out Two K_ Transporters in the Euascomycete Fungus Podospora anserine. *Genetics*, 2004, vol. 166, p. 125-133. PMid:15020412. http://dx.doi.org/10.1534/genetics.166.1.125

MEGENEY, LA and RUDNICKI, MA. Determination versus differentiation and the MyoD family of transcription factors. *Biochemistry & Cell Biology*, 1995, vol. 73, n. 9-10, p. 723-732. PMid:8714693. http://dx.doi.org/10.1139/o95-080

MOORE, KL. and DALLEY, AF. *Anatomia Orientada para Clínica*. Rio de Janeiro: Guanabara Koogan, 2007.

MORAIS, MA., GOMES, MS., HELRIGLE, C and MALYSZ, T. Prevalence of agenesis of the palmaris longus muscle in Brazil and its clinics correlation. *Journal of Morphological Sciences*, 2012, vol. 29, n. 4, p. 1-5.

MUNTONI, F., BROWN, S., SEWRY, C. and PATEL, K. Muscle development genes: their relevance in neuromuscular disorders. *Neuromuscular Disorders*, 2002, vol. 12, n. 5, p. 438-446. http://dx.doi.org/10.1016/S0960-8966(01)00326-1

MURRE, C., McCAW, PS., VAESSIN, H., CAUDY, M., JAN, LY., JAN, YN., CABRERA, CV., BUSKIN, JN., HAUSCHKA, SD., LASSAR, AB., WEINTRAUB, H. and BALTIMORE, D. Interactions between heterologous helix-loop-helix proteins generate complexes that bind specifically to a common DNA sequence. *Cell,* 1989, vol. 58, n. 3, p. 537-544. http://dx.doi.org/10.1016/0092-8674(89)90434-0

NAIDU, PS., LUDOLPH, DC., TO, RQ., HINTERBERGER, TJ. and KONIECZNY, SF. Myogenin and MEF2 function synergistically to activate the MRF4 promoter during myogenesis. *Molecular and Cellular Biology*, 1995, vol. 15, n. 5, p. 2707-2718. PMid:7739551 PMCid:PMC230501.

NAUGLE, TCJR. and FAUST, DC. Autogeneous Palmaris longus tendon as frontalis suspension material for ptosis correction in children. *American Journal of Ophthalmology*, 1999, vol. 127, n. 4, p. 488-9. http://dx.doi.org/10.1016/S0002-9394(99)00047-1

NAYA, FJ. and OLSON, E. MEF2: A transcriptional target for signaling pathways controlling skeletal muscle growth and differentiation. *Current opinion in cell biology*, 1999, vol. 11, n. 6, p. 683-688. http://dx.doi.org/10.1016/S0955-0674(99)00036-8

O'SULLIVAN, E. and MITCHELL, BS. Association of the absence of the Palmaris longus tendon with an anomalous superficial palmar arch in the human hand. *Journal of Anatomy*, 2003, vol. 201, n. 5, p. 405-408. http://dx.doi.org/10.1046/j.0021-8782.2002.00109.x

PEREIRA, L., D'ALESSIO, M., RAMIREZ, F., LYNCH, JR., SYKES, B., PANGILINAN, T. and BONADIO, J. Genomic organization of the sequence coding for fibrillin, the defective gene product in Marfan syndrome. *Human Molecular Genetics*, 1993, vol. 2, n. 7, p. 961-968. PMid:8364578. http://dx.doi.org/10.1093/hmg/2.7.961

REGATEIRO, FJ. *Manual de Genética Médica*. Coimbra: Imprensa da Universidade de Coimbra, 2007. 496 p.

SCHAEFFER, JP. On the variations of the palmaris longus muscle. *Anatomical Record,* 1909, vol. 3, p. 275-278.

SEBASTIN, SJ., PUHAINDRAN, ME., LIM, AY., LIM, IJ. and BEE, WH. The prevalence of absence of the palmaris longus - A study in a Chinese population and a review of the literature. *Journal of Hand Surgery*, 2005, vol. 30, n. 5, p. 525-7.

TAJBAKHSH, S., ROCANCOURT, D., COSSU, G. and BUCKINGHAM, M. 1997. Redefining the genetic hierarchies controlling skeletal myogenesis: PAX-3 and Myf-5 act upstream of MyoD. *Cell*, 1997, vol. 89, n. 1, p. 127-138. http://dx.doi.org/10.1016/S0092-8674(00)80189-0

THOMPSON, JW., MCBATTS, J. and DANFORTH, CH. 1921. Hereditary and racial variations in the musculus palmaris longus. *American Journal of Physical Anthropology*, 1921, vol. 4, n. 11, p. 205-220.

TROHA, F., BAIBAK, GJ. and KELLEHER, JC. Frequency of the palmaris longus tendon in North American Caucasians. *Annals of Plastic Surgery*, 1990, vol. 25, n. 6, p. 477-478. PMid:2073079. http://dx.doi.org/10.1097/00000637-199012000-00008

VANDERHOOFT, E. 1996. The frequency and relationship between the palmaris longus and plantaris tendons. *American Journal of Orthopedics*, 1996, vol. 25, n. 1, p. 38-41. PMid:8722127.

WEHBE, MA. Tendon graft donor sites. *Journal of Hand Surgery*, 1992, vol. 17, n. 6, p. 1130-2. http://dx.doi.org/10.1016/S0363-5023(09)91079-6

YILDIZ, N., SNER, M. and AYNACI, O. Three-headed reversed Palmaris longus muscle: a report and review of the literature. *Surgical and Radiologic Anatomy*, 2000, vol. 22, n. 3-4, p. 217-219. PMid:11143317. http://dx.doi.org/10.1007/s00276-000-0217-x

Received March 6, 2013
Accepted December 17, 2013