Expression of MuRF1 and MAFbx in Donor and Recipient Muscles after Musculocutaneous Nerve Transection and Partial Pectoralis Major Muscle Transfer for Reconstruction of Elbow Flexion in Rats

Expresión de MuRF1 y MAFbx en Músculos Donantes y Receptores después de Transección del Nervio Musculocutáneo y Transferencia Parcial del Músculo Pectoral Mayor para la Reconstrucción de Flexión de Codo en Ratas

Shengbo Yang*; Ning Yue* & Xueqing Zeng**


SUMMARY: The expression of MuRF1 and MAFbx in a denervated muscle has previously been studied. However, the expression of MuRF1 and MAFbx in the recipient and donor muscles after muscle transfer for reconstruction of joint function has not been sufficiently investigated. Forty-two adult Sprague-Dawley rats were divided into 7 groups: normal, 1 w post-, 2 w post-, and 4 w post-musculocutaneous nerve transection; and 1 w post-, 2 w post-, and 4 w post-reconstruction of elbow flexion. Muscle wet weights were assessed, and MuRF1 and MAFbx mRNA expressions were detected by polymerase chain reaction. The length of the oblique part of the pectoralis major of an SD rat is sufficient for suture to the insertion of the biceps brachii tendon. The muscle wet weight and the wet weight retention rate of the biceps brachii continued to decline after musculocutaneous nerve transection and a gradual increase was noted after the oblique part of the pectoralis major was transferred for reconstruction of elbow flexion. The oblique part of the pectoralis major showed a decrease of only 2–6%. The upregulated expression of MuRF1 and MAFbx in the biceps brachii reached a peak 2 w after denervation and 1 w after elbow flexion reconstruction, with an increase of 15% and 4%, respectively. This was followed by downregulation; however, the expression had not normalized at postoperative 4 w. The increased expression of MuRF1 (17%) and MAFbx (1%) in the oblique part of the pectoralis major at postoperative 1 w had decreased to below normal levels at postoperative 4 w. The transfer of the oblique part of the pectoralis major for elbow flexion reconstruction after musculocutaneous nerve transection can downregulate the expression of MuRF1 and MAFbx in the recipient muscle and causes only transient damage to the donor muscle in rats.

KEY WORDS: Musculocutaneous nerve injury; MuRF1; MAFbx; Pectoralis major; Biceps Brachii.

INTRODUCTION

A musculocutaneous nerve injury can result from an open injury such as a cut, a pull injury, joint dislocation or fracture displacement, and baseball and softball sports injuries (Hsu et al., 2007; DeFranco & Schickendantz, 2008). Clinically, in a musculocutaneous nerve injury that occurred more than 2 years previously or one in which the musculocutaneous nerve is repaired after one year, no functional recovery is noted, the biceps brachii and brachialis muscles are atrophied, and elbow flexion reconstruction is required as the brachioradialis muscle cannot compensate fully for elbow flexion. Furthermore, the pectoralis major muscle can be divided into 5 parts: the clavicular, manubrial, sternocostal, costal, and abdominal portions. For treatment in the above cases, the origins of the sternocostal, costal, and abdominal parts of the pectoralis major are cut off as a unit, shifted, and then sutured to the biceps tendon insertion. The muscle fiber length and strength of the 3 parts are similar to those of the biceps brachii; therefore, this is an ideal material for functional reconstruction of the elbow (Atkins et al., 1985; Chomiak & Dungl, 2008).

The expressions of the muscle-specific RING-finger protein 1 (MuRF1) and muscle atrophy F-box (MAFbx) mRNA of denervated skeletal muscle have been reported in
the literature (Moriscot et al., 2010; Goncalves et al., 2012). However, their expression in the donor and recipient muscles after muscle transplantation has not been sufficiently studied. After muscle transplantation, greater attention is paid to the macroscopic changes in the donor and recipient muscles, to assess if they are alive, and to the functional recovery; thus, the changes at the molecular level are often ignored. Therefore, in the present study, we aimed to detect the changes in the expression of MuRF1 and MAFbx mRNA in the recipient and donor muscles after transection of the musculocutaneous nerve and partial transplantation of the pectoralis major for reconstruction of elbow flexion in rats, and to provide clinically appropriate measures for the prevention and treatment of postoperative muscle atrophy.

MATERIAL AND METHOD

Animal Care and Ethics Statement. Forty-two adult Sprague Dawley rats (mean weight, 200±50 g) were housed in individual cages and fed standard rat chow and water ad libitum. The animals were divided into 7 groups: normal, 1 w post-, 2 w post-, and 4 w post-musculocutaneous transection; and 1 w post-, 2 w post-, and 4 w post-reconstruction of elbow flexion. Each group comprised 6 rats. All experiments were performed in accordance with the guidelines of the China Animal Welfare Act. This study was approved by the Animal Care and Use Committee of Zunyi Medical College. All the surgical steps were conducted strictly in accordance with the aseptic principles of surgery set forth by Zunyi Medical College.

Gross Anatomy. The rats were sacrificed by CO2 inhalation followed by decapitation. The morphology, color, and entry point of the blood vessels and nerves of the recipient muscles (biceps brachii) and donor muscles (pectoralis major) were studied based on gross anatomy. The origin of the oblique part of the pectoralis major, which is roughly equivalent to the size of the biceps was cut off, separated, inverted on the biceps brachii insertion, and sewn on the biceps brachii insertion according to the available length. Precautions were taken to prevent damage to blood vessels and nerves.

Surgery. The rats were anesthetized by an intraperitoneal injection of 0.5 mL/100 g 4% chloral hydrate. The superior aspect of the right forelimb and right chest were disinfected with an iodophor disinfectant. A layer-by-layer incision was made; the musculocutaneous nerve stem was freed and resected (0.5 cm), followed by layer-by-layer suturing (Fig. 1C). The rats were then housed in individual cages; they were divided into 3 groups and fed standard rat chow and water ad libitum for 1, 2, and 4 w, respectively. The SD rats in which the musculocutaneous nerves had been transected 4 w previously were anesthetized again, followed by regular disinfection and a “T” incision. The oblique part of the pectoralis major, equivalent to the size of the biceps brachii, was used as the donor. It was shifted and sutured to the biceps tendon insertion for restoration of elbow flexion (Fig. 1D). The rats were then housed in individual cages; they were divided into 3 groups and fed standard rat chow and water ad libitum for 1, 2, and 4 w, respectively (i.e. 5, 6, and 8 w after the transection of the musculocutaneous nerve, respectively).

Observation and Measurement. The foraging movements and gait of each animal were observed carefully. The muscle block size and color of the biceps brachii were observed at all time points after denervation and those of the oblique part of the pectoralis major were observed at all time points after reconstruction of elbow flexion. The right and left biceps brachii and oblique part of the pectoralis major were weighed at each time point, and the muscle wet weight retention was calculated by dividing the muscle wet weight of the injured side (right) by the muscle wet weight of the normal side (left) and multiplying by 100% (Liu et al., 2009).

Reverse transcriptase-polymerase chain reaction (RT-PCR). Total RNA was extracted from the biceps brachii and oblique part of the pectoralis major in SD rats using the TRIzol reagent (Invitrogen, USA). One microliter of total RNA was used for reverse transcription reaction using the ReverTraid First Strand cDNA Synthesis Kit (Thermo Scientific, USA). Thereafter, the DreamTaq Green PCR Master Mix (2X) kit (Thermo Scientific, USA) was used for PCR amplification; the PCR electrophoresis bands were photographed in the gel imager, and the gray values of the bands were measured by IPP6.0 software. The gray value of each objective gene band divided by the gray value of GAPDH represents the mRNA expression level. The sequences of MuRF1, MAFbx, and GAPDH primers according to the design and synthesis of Shanghai Biological Engineering Co. Ltd are as follows:

MuRF1-F: 5¢-CTACAAGCAGAATGCTGCTCG-3¢; MuRF1-R: 5¢-GAGGGCCTCAAAACTTGTG-3¢; MAFbx-F: 5¢-CCTCACACTGGGCAACAGCA-3¢; MAFbx-R: 5¢-CGCTCTGAGAAGTGGTACTGG-3¢; GAPDH-F: 5¢-TCCGTGCCACCAAAGGTGACTTACRGG-3¢; GAPDH-R: 5¢-TAGGGAGATTGCCCTTATGATGG-3¢.

Scheme of the experimental setup.
Statistical Processing. Data were analyzed by one-way ANOVA, with a P value <0.05 being considered statistically significant.

RESULTS

Gross Observations. In Sprague Dawley rats, the musculocutaneous nerve and artery of the biceps brachii enter the superior part of the muscle belly from its medial edge, and the lateral pectoral nerve and lateral thoracic artery of the pectoralis major enter the muscle insertion from the profundal face of the muscle (Fig. 1A and 1B). Based on the direction of the muscle fibers, the pectoralis major can be divided into 2 parts: transverse and oblique. The length of the oblique part of the pectoralis major is sufficient for suture to the biceps brachii tendon (Fig. 1D). After the musculocutaneous nerve transection, the color of the biceps brachii muscle faded progressively and the muscle mass shrank (Fig. 2A–D). After reconstruction of elbow flexion, the muscle turned red and shiny and the muscle mass increased slightly (Fig. 2E–G). The oblique part of the pectoralis major showed minor changes postoperatively. After musculocutaneous nerve transection, the limp in the rats worsened and the foraging movements were restricted. One week after transplantation of the oblique part of the pectoralis major, the limp and foraging action improved, although they had not normalized at 4 w.

Muscle Wet Weight and Wet Weight Retention Rate. The muscle wet weight and wet weight retention rate of the biceps brachii and oblique part of the pectoralis major (transplanted) were assessed at each time point (Tables I and II). The normal muscle wet weight of the biceps brachii and oblique part of the pectoralis major were $0.31 \pm 0.06$ g and $0.32 \pm 0.02$ g, respectively. The muscle wet weight of the biceps brachii decreased by 0.12, 0.14, and 0.19 g at 1, 2, and 4 w, respectively, after musculocutaneous nerve transection. The muscle wet weight of the biceps brachii showed an increase of 0.06, 0.07 and 0.07 g compared to the normal weight at 1, 2, and 4 w after the transfer for elbow flexion reconstruction ($P=0.036$). The wet weight of the oblique part of the pectoralis major decreased by 0.05, 0.03, and 0.02 g compared to normal at 1, 2, and 4 w, respectively, after musculocutaneous nerve transection. The muscle wet weight of the biceps brachii showed an increase of 0.06, 0.07 and 0.07 g compared to the normal weight at 1, 2, and 4 w after the transfer for elbow flexion reconstruction ($P=0.036$). The wet weight of the oblique part of the pectoralis major decreased by 0.05, 0.03, and 0.02 g compared to normal at 1, 2, and 4 w, respectively, after the transfer (1 w, $P=0.034$; 2 w and 4 w, $P=0.037$). At 1, 2, 4, 5, 6, and 8 w, the muscle wet weight retention rates of the biceps brachii were 70%, 55%, 42%, 65%, 59%, and 61%, respectively, compared to that of the normal group ($P=0.039$). At 1, 2, and 4 w after the elbow flexion reconstruction.
Table I. Wet weight of biceps brachii and oblique part of the pectoralis major at different time points in rats (n=6, Mean±SD).

<table>
<thead>
<tr>
<th>Group</th>
<th>Biceps brachii</th>
<th>Oblique part of pectoralis major</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Right (g)</td>
<td>Left (g)</td>
</tr>
<tr>
<td>Normal (0 w)</td>
<td>0.31±0.06</td>
<td>0.31±0.03</td>
</tr>
<tr>
<td>1 w post-musculocutaneous nerve transection</td>
<td>0.19±0.05</td>
<td>0.26±0.04</td>
</tr>
<tr>
<td>2 w post-musculocutaneous nerve transection</td>
<td>0.17±0.04</td>
<td>0.33±0.07</td>
</tr>
<tr>
<td>4 w post-musculocutaneous nerve transection</td>
<td>0.12±0.02</td>
<td>0.29±0.06</td>
</tr>
<tr>
<td>1 w post-reconstruction of elbow flexion</td>
<td>0.18±0.04</td>
<td>0.28±0.05</td>
</tr>
<tr>
<td>2 w post-reconstruction of elbow flexion</td>
<td>0.19±0.03</td>
<td>0.33±0.05</td>
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</table>

Table II. Wet weight retention rate of the biceps brachii and oblique part of the pectoralis major on the operated side in rats (n=6, Mean±SD).

<table>
<thead>
<tr>
<th>Group</th>
<th>Biceps brachii</th>
<th>Oblique part of pectoralis major</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>(%)</td>
<td>(%)</td>
</tr>
<tr>
<td>Normal (0 w)</td>
<td>100.00±2.68</td>
<td>100.00±3.00</td>
</tr>
<tr>
<td>1 w post-musculocutaneous nerve transection</td>
<td>70.02±5.08</td>
<td>100.00±3.01</td>
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<tr>
<td>2 w post-musculocutaneous nerve transection</td>
<td>55.01±4.77</td>
<td>100.00±3.03</td>
</tr>
<tr>
<td>4 w post-musculocutaneous nerve transection</td>
<td>42.04±2.03</td>
<td>100.00±3.06</td>
</tr>
<tr>
<td>1 w post-reconstruction of elbow flexion</td>
<td>65.06±4.10</td>
<td>94.00±6.02</td>
</tr>
<tr>
<td>2 w post-reconstruction of elbow flexion</td>
<td>59.05±3.01</td>
<td>98.00±2.07</td>
</tr>
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</table>

Table III. Expression level (indicated in grey) of MuRF1 and MAFbx of biceps brachii and oblique part of the pectoralis major at different time points in rats (n=6, Mean±SD).

<table>
<thead>
<tr>
<th>Group</th>
<th>Biceps brachii</th>
<th>Oblique part of pectoralis major</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>MuRF1</td>
<td>MAFbx</td>
</tr>
<tr>
<td></td>
<td>MuRF1</td>
<td>MAFbx</td>
</tr>
<tr>
<td>Normal (0 w)</td>
<td>0.78±0.003</td>
<td>0.76±0.003</td>
</tr>
<tr>
<td>1 w post-musculocutaneous nerve transection</td>
<td>0.92±0.005</td>
<td>0.87±0.007</td>
</tr>
<tr>
<td>2 w post-musculocutaneous nerve transection</td>
<td>0.94±0.004</td>
<td>0.89±0.007</td>
</tr>
<tr>
<td>4 w post-musculocutaneous nerve transection</td>
<td>0.83±0.006</td>
<td>0.78±0.008</td>
</tr>
<tr>
<td>1 w post-reconstruction of elbow flexion</td>
<td>0.90±0.007</td>
<td>0.79±0.009</td>
</tr>
<tr>
<td>2 w post-reconstruction of elbow flexion</td>
<td>0.81±0.006</td>
<td>0.77±0.008</td>
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Fig. 3. The changes in the expression of MuRF1 and MAFbx of donor and recipient muscles after transection of the musculocutaneous nerve and partial pectoralis major muscle transfer for reconstruction of elbow flexion in SD rats. A and C show results of polymerase chain reaction electrophoresis for MuRF1 and MAFbx in the biceps brachii at different time points. M= marker; 1= lane for glyceraldehyde-3-phosphate dehydrogenase (GAPDH); 2= lane for normal control (0 w); 3, 4, 5= lanes for 1, 2, and 4 w after the transection of the musculocutaneous nerve, respectively; 6, 7, 8= lanes for 5, 6, and 8 w after the musculocutaneous nerve transection, respectively (i.e., 1, 2, and 4 w after transplantation of the oblique part of the pectoralis major, respectively). B and D show results of PCR electrophoresis for MuRF1 and MAFbx the oblique part of the pectoralis major at different time points. M= marker; 1= lane for GAPDH; 2= lane for normal group (0 w); 3, 4, 5= lanes for 1, 2, and 4 w after transplantation of the oblique part of the pectoralis major, respectively (i.e., 5, 6, and 8 w after musculocutaneous nerve transection, respectively).
reconstruction, the muscle wet weight retention rates of the oblique part of the pectoralis major were 94%, 98% and 98%, respectively (at 1 w, P= 0.038; at 2 and 4 w, P= 0.072).

Expression of MuRF1 and MAFbx mRNA. The MuRF1 and MAFbx mRNA expression levels in the biceps brachii and oblique part of the pectoralis major at each time point were measured (Table III, Figs. 3A–D and 4). MuRF1 and MAFbx expression in the denervated biceps brachii muscle was increased by 18% and 15%, respectively, at 1 w and was upregulated to the peak value of 21% and 17%, respectively, at 2 w. After 2 w, the expression began to decrease; at 4 w, it was only 6% and 3% more than normal for MuRF1 and MAFbx, respectively. One week after transfer of the oblique part of the pectoralis major for restoration of elbow flexion, the expression of MuRF1 and MAFbx in the biceps brachii had increased by 15% and 4%, respectively, followed by a downregulation to 4% and 1% more than the normal at 2 w; at 4 w, the expression had still not normalized (compared to the normal, P= 0.035, power = 0.93). One week after transfer of the oblique part of the pectoralis major for restoration of elbow flexion, its MuRF1 and MAFbx mRNA expression increased by 17% and 1%, respectively; at 2 w, the expression showed a rapid decrease, and at 4 w, it decreased to 13% below normal and 4% below normal, respectively (P= 0.040, power= 0.93). The changes in the expression of MuRF1 was greater than that of MAFbx at different time points in Sprague Dawley rats (Fig. 4).

DISCUSSION

Skeletal muscle atrophy can occur in many diseases and conditions, such as denervation, cancer cachexia, aging, sepsis, diabetes, renal failure, major trauma, infections, hyperthyroidism, and weightlessness (Bodine et al., 2001). MuRF1 and MAFbx are ubiquitin ligases that are required for skeletal muscle atrophy, and are markers of skeletal muscle atrophy (Ogawa et al., 2006; Tisdale, 2007; Jones et al., 2010; Labeit et al., 2010; Meinen et al., 2012; Bentzinger et al., 2013; Ogawa et al., 2013). Rowan et al., used adult rats and aged rats for a comparative study and found that the change in the patterns of MAFbx and MuRF1 expression in denervated muscle were similar to those noted in the denervation-specific sodium channel positive fibers that had higher levels of MAFbx and MuRF1 and smaller sized muscle fibers in aged rats. Furthermore, their expression ratio in aged rats was higher than that in the adult rats, probably because aged rats in vivo have experienced age-related denervation. This finding indicates that denervation is an important cause of skeletal muscle atrophy (Rowan et al., 2010).

In Sprague Dawley rats, the musculocutaneous nerve and artery of the biceps brachii enter the superior part of the muscle belly from its medial edge, and the lateral pectoral nerve and lateral thoracic artery of the pectoralis major enter the muscle insertion from the profundal face of the muscle. The pectoralis muscle can be divided into the transverse and oblique parts based on the direction of its muscle fibers. The length of the oblique part is sufficient for suture to the biceps brachii tendon. These findings suggest that there is no risk of injury to the blood vessels and nerves during transfer of the oblique part of the pectoralis major muscle for reconstruction of elbow flexion in an animal model. One to four weeks after musculocutaneous nerve injury, claudication, limited foraging, and color fading and muscle mass shrinkage of the biceps brachii were observed. These are undoubtedly the signs of neurogenic muscular atrophy. One to four weeks after transfer of the oblique part of the pectoralis major muscle for reconstruction of elbow flexion, the color of the biceps brachii gradually turned red and the muscle mass recovered slightly. We believe that these changes were due to donor muscle contraction that led to the passive movement of the recipient muscle. However, the color and muscle size of the oblique part of the pectoralis major showed only a mild change postoperatively, indicating that the surgery caused almost nodamage to the donor muscle.

Zeman et al. (2009), have reported that after sciatic nerve axotomy, the expression of MuRF1 and MAFbx mRNA in the extensor digitorum longus was the highest at 3 days and the lowest at 1 w; the expression slowly increased...
again between 1 and 2 w, suggesting that atrophy of the extensor digitorum longus improves gradually in the 1–2 w period. The MuRF1 and MAFbx mRNA expression in the soleus muscle was also highest at 3 days, and decreased progressively in the later stage, suggesting that soleus muscle atrophy decreased gradually after 3 days. These results suggest that the degree of muscle atrophy following denervation differs between muscles and between different time points (Zeman et al.). Our experimental results (Tables I and II) show that the muscle wet weight and muscle wet weight retention of the biceps brachii decreased gradually at 1, 2, and 4 w after musculocutaneous nerve injury, and showed an increase after 5, 6, and 8 w (i.e., at 1, 2, and 4 w after the reconstruction of elbow flexion, respectively). These results illustrate that contraction of the transplanted pectoralis major causes passive contraction and movement of the biceps brachii, thereby improving the muscle atrophy of the biceps brachii.

The MuRF1 and MAFbx mRNA expression in the denervated gastrocnemius muscle of mice was increased markedly, along with obvious muscle atrophy at 4 w (Mittal et al., 2010). One hind limb of the rat was selected to receive in situ free gracilis transplantation; at 2 weeks, the MAFbx/atrogin-1 mRNA expression increased 7 times compared to that in the contralateral side and continued to increase at 4–15 weeks with progressive muscular atrophy. At 15–30 weeks, the expression decreased gradually, and muscle atrophy improved (Liu et al., 2009). In our results (Table III, Figs. 3A–D and 4), a progressive upregulation of MuRF1 and MAFbx mRNA expression was observed in the biceps brachii at 1–2 weeks after musculocutaneous nerve injury. The expression peaked at 2 w, after which it decreased, and reached near normal levels at 4 w. This shows that the muscle atrophy gradually increased during the 1–2 w period; the increase in atrophy was greatest at 2 w, after which the atrophy decreased, and a mild state of atrophy was noted at 4 w. This change in the pattern noted in the present study was in agreement with that noted in Zeman’s study (Zeman et al.). After the oblique part of the pectoralis major was transplanted, the expression change rate of MuRF1 mRNA in the biceps brachii increased at first and then decreased; however, at 8 w, the levels were still above the normal level. The suture-induced minor damage to the biceps brachii, may have caused the initial increase in the expression. Downregulation indicates contraction of the transplanted pectoralis major caused by passive contraction of the biceps brachii; however, since the biceps brachii were denervated, expression of MuRF1 and MAFbx mRNA were not reduced to normal levels. After 1 week of transfer of the oblique part of the pectoralis major for elbow flexion reconstruction, increased expression of MuRF1 and MAFbx mRNA was noted in the oblique part of the pectoralis major, which then decreased rapidly to below normal at 2 w. We believe that this increased expression was induced by cutting and displacement of the oblique part of the pectoralis major origin. At 2 w, because this oblique part was sutured to the biceps brachii tendon, it contracted; therefore, the expression decreased rapidly. We speculate that the expression subsequently decreased to below normal because the position of the oblique muscle fibers and the force direction changed, resulting in greater contraction amplitude and activity of the oblique muscle fibers. Therefore, we believe that the oblique part of the pectoralis major transplant can not only ensure recovery of elbow flexion but also improve the recipient muscle atrophy; in such cases, the donor muscle itself undergoes only a mild injury that lasts for 1 w after the surgery. In addition, the expression change rate of MuRF1 mRNA in the denervated recipient muscle (biceps brachii) and donor muscle (oblique part of the pectoralis major) is greater than that of the MAFbx. This result suggests that the sensitivity of expression of the 2 ubiquitin ligases is different and may be regulated by different proteolysis mechanisms. Based on the above findings, we suggest that in order to prevent and provide better treatment for donor and recipient muscle atrophy associated with this surgery, physicians should select the appropriate inhibitors based on the timing of the atrophy and the expression rate of MuRF1 and MAFbx.

In conclusion, The transfer of the oblique part of the pectoralis major for elbow flexion reconstruction after musculocutaneous nerve transection can downregulate the expression of MuRF1 and MAFbx in the recipient muscle and causes only transient damage to the donor muscle in rats. The expression change rate of MuRF1 mRNA in the denervated recipient muscle (biceps brachii) and donor muscle (oblique part of the pectoralis major) is greater than that of the MAFbx at different time points in rats.

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7 grupos: normales, 1 semana post-, 2 semanas post- y 4 semanas post-transsección del nervio musculocutáneo; y 1 semana post-, 2 semanas post-, y 4 semanas post-reconstrucción de la flexión del codo. Se evaluó el peso de los músculos húmedos, y las expresiones de MuRF1 y MAFbx mRNA fueron detectadas a través de reacción en cadena de la polimerasa. La longitud de la parte oblicua del músculo pectoral mayor de una rata Sprague-Dawley es suficiente para realizar la sutura en la inserción del tendón de músculo bíceps braquial. El peso húmedo del músculo bíceps braquial y su tasa de retención siguieron disminuyendo después de la sección del nervio musculocutáneo y un aumento gradual se observó después de la transferencia de la parte oblicua del músculo pectoral mayor para la reconstrucción de la flexión del codo. La parte oblicua del músculo pectoral mayor mostró una disminución de sólo 2-6%. La expresión regulada por incremento de MuRF1 y MAFbx en el bíceps braquial alcanzó un peak 2 semanas después de la denervación y 1 semana después de la reconstrucción de la flexión del codo, con un incremento del 15% y el 4%, respectivamente. Esto fue seguido por un regulación en baja. Sin embargo, la expresión no se normalizó en el postoperatorio de las 4 semanas. El aumento de la expresión de MuRF1 (17%) y MAFbx (1%) en la parte oblicua del músculo pectoral fue mayor en el postoperatorio de 1 semana, mientras que se encontró por debajo de los niveles normales en el postoperatorio de 4 semanas. La transferencia de la parte oblicua del músculo pectoral mayor para la reconstrucción de la flexión del codo después de la sección del nervio musculocutáneo puede regular a la baja la expresión de MuRF1 y MAFbx en el músculo receptor y provocar solo un daño transitorio en el músculo donado en ratas.

PALABRAS CLAVE: Lesión del nervio musculocutáneo; MuRF1; MAFbx; Músculo pectoral mayor; Músculo bíceps braquial.

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