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Variation in isometric force after active shortening and lengthening and their mechanisms: a review

Variação da força isométrica após encurtamento e alongamento ativo e seus mecanismos: uma revisão

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Abstract

Introduction: The isometric force history dependence of skeletal muscle has been studied along the last one hundred years. Several theories have been formulated to explain and establish the causes of the phenomenon, but not successfully, as they have not been fully accepted and demonstrated, and much controversy on such a subject still remains. **Objective**: To present a systematic literature review on the dynamics of the mechanisms of force depression and force enhancement after active shortening and lengthening, respectively, identifying the key variables involved in the phenomenon, and to date to present the main theories and hypothesis developed trying to explaining it. **Method**: The procedure of literature searching complied the major databases, including articles either, those which directly investigated the phenomena of force depression and force enhancement or those which presented possible causes and mechanisms associated with their respective events, from the earliest studies published until the year of 2010. **Results**: 97

references were found according to the criteria used. **Conclusion**: Based on this review, it is suggested that the theory of stress inhibition of actin-myosin cross-bridges is that better explain the phenomenon of force depression. Whereas regarding the force enhancement phenomenon, one theory have been well accepted, the increased number of actin-myosin cross-bridges in strong binding state influenced by the recruitment of passive elastic components, which hole is attributed to the titin filament.

Keywords: Force. Shortening. Lengthening. Active.

Resumo

Introdução*: A dependência entre a produção de força isométrica do músculo esquelético e o histórico da contração precedente vem sendo objeto de estudo nos últimos cem anos. Diversas teorias têm sido formuladas para explicar e estabelecer as causas do fenômeno, obtendo pouco sucesso, uma vez que não foram completamente aceitas e comprovadas, suscitando ainda muita controvérsia sobre o tema.* ***Objetivos****: Apresentar uma revisão sistemática da literatura sobre a dinâmica dos mecanismos de depressão (DF) e de aumento (AF) da força isométrica após encurtamento e alongamento ativos, respectivamente, identificando as principais variáveis intervenientes nos fenômenos, e as principais teorias e hipóteses elaboradas para explicá-los até a presente data.* ***Métodos****: O procedimento de busca da literatura foi composto por uma pesquisa nas principais bases de dados, incluindo artigos que observaram diretamente os fenômenos DF e AF, ou possíveis mecanismos e causas associadas a suas respectivas manifestações, desde os primeiros estudos sobre o tema até o ano de 2010.* ***Resultados****: Foram encontradas 97 referências que atenderam aos critérios adotados.* ***Conclusão****: Com base na revisão produzida, sugere-se a teoria da inibição por estresse induzido das pontes cruzadas de actina-miosina para explicar o fenômeno DF. Quanto ao fenômeno AF uma teoria tem sido bem aceita, a do aumento do número de pontes cruzadas de actina-miosina em estado forte de ligação influenciado pelo recrutamento dos componentes elásticos passivos, atribuindo-se aos filamentos de titina este papel.*

Palavras-chave*: Força. Encurtamento. Alongamento. Ativo.*

Introduction

The dependence between the production of isometric force in skeletal muscle and its contraction history has been reported nearly a century ago (1, 2). However, its causes are not well established, and the proposed theories not fully accepted, as well as controversial. It is relevant that such dependence is not taken into account by the models of Hill and Huxley, the two biomechanical models that try to explain the dynamics of mechanics and physiology of muscle contraction (3, 4). Moreover, despite the scientific advances in the field of muscle mechanics, a great difficulty is remains while accepting new discoveries and theories of force depression (FD) and force increased (FE) (5, 6, 7).

Studies have shown that the isometric force of a sarcomere, a fiber or even a muscle, may change up to 50% when compared to a known isometric force of reference, being reduced after active shortening and increased after stretching, phenomena know as FD and FE respectively (7) .

A concept well accepted is that force capacity of a muscle force has an inverse non-linear relationship with the speed of shortening (2, 3, 8, 9, 10, 11, 12, 13). In an antagonist way, the capacity of power generation increases due to the speed of stretching to a limited extent, mainly when the muscle is stimulated beyond its optimal length (2, 3, 9, 10, 13). These characteristics of plasticity and strength of a muscle when taken as a principle after systematic experimental observations has helped to develop the concepts associated with FD and FE (1, 2, 11).

Until recently, little was known about the occurrence of FE and FD in large muscle groups during movements with production of submaximal and voluntary force (5). That is because most of studies on this topic used sarcomeres, muscle fibers of animal specimens, or small muscle groups such as the adductors of the thumb, in conditions of electrical

stimulation combined with maximum voluntary dynamic contractions (5). Furthermore, devices have relied on complex servo motors coupled to force transducers that bear little resemblance to human movement pattern (2, 11, 14, 15, 16, 17, 18, 19, 20, 21, 22, 23, 24, 25, 26).

Moreover, Herzog (7) proposed that FD and FE might have implications in the tasks of daily life. This phenomenon, once elucidated, could also bring important information when regarded to rehabilitation processes, in which small increases in strength capability are crucial to patient outcomes.

Another important aspect is that recent studies have observed the manifestation of FD and FE in higher muscle groups and motor gestures more similar to human routine, both in maximal voluntary and submaximal force exertions (27, 28, 29, 30, 31). It is noteworthy that although there are many studies proving the existence of FD and FE as well as some hypotheses to explain such behaviors has been raised, no theory is fully accepted to explain such behaviors.

In fact, there are many variables involved in the change of intensity force produced or even on whether or not this change occurs (6, 7, 32). Such variables include the amount of stimulation, speed and length vs muscle tension curve (LTC) region. Furthermore, there are gaps in these phenomena with regard to its applicability to daily life, despite being touted as important in motor control with implications in rehabilitation (5, 7).

This paper aims to present a systematic review on the concepts related to depression and enhancement of isometric strength after active stretching and shortening respectively. It also intends to describe the main variables that affect the phenomena. Additionally, the main mechanisms, theories and hypotheses to explain them are presented.

Employed procedures and methods of search

This article consists of a systematic literature review on the topics known as FD and FE. The procedure consisted in literature search in major databases (PubMed, Medline, SciELO, SPORTDiscus, LILACS and Academic Google), including the first records on the subject until the year 2010.

The following keywords were used for the searches: isometric force, speed of shortening, speed of stretching, force depression, force enhancement, residual force enhancement, passive elastic component, history dependence of muscle contraction, sarcomere length, shortening, lengthening, stretch- shortening cycle, stretch - shortening cycle, calcium dependent mechanism of muscle contraction, titin filament and thin. The words were used alone or combined.

To be included in the review, articles should address the observation of the phenomena of FD or FE, alone or combined, as well as present description of possible causes or physiological and biomechanical mechanisms involved in its manifestation. Were also included articles that presented theories on the mechanics of muscle contraction and that are relevant to the topic. In this search, 97 references met the inclusion criteria.

The concept of Force Depression

The term FD can be defined as the reduction of isometric force after active muscle shortening when compared to a reference isometric contraction obtained in the same muscle length. In the first case, the phenomenon would be associated with reduced state of passive tension of muscle fibers, individually, during the shortening phase (6, 7, 33, 34).

It is proposed that the phenomenon of FD would associated to the descending limb and to the peak of the SLT (35, 36, 37), but recent evidence suggests that this would happen too, even on a smaller scale, the ascending limb of the LTC (21, 32, 38, 39).

Literature data point to the idea that the intensity of the decline of isometric force is directly influenced by the extent of muscle shortening, the shortening speed and the amount of force produced during the shortening phase (2, 6, 7, 14, 15, 16, 33). This can be seen in the summary of the studies shown in Table 1, which also shows that the employed force control, in most studies, is made by frequency of the stimulation produced in the sarcomere, or to muscle fiber.

Relevant fact is that the phenomenon has a greater duration than five seconds (2, 6, 33, 40, 41), considered expressive by the investigator of such subject. Moreover, the FD cannot be completely eliminated after a maneuver of muscle relaxation by deactivation nor manipulated by shortening with fast speed, preceded by a shortening with slow and steady speed, which would result in a reduction of the isometric force to zero (6, 7, 15, 42).

However, it should be noted that, considering the application of stretching *vs* shortening cycle the phenomenon of FD may be entirely abolished only if the previous amount of stretching is equivalent to the amount of shortening. Otherwise, the isometric force produced after deactivation maneuver will always be less than the isometric reference force (2, 6, 7, 33).

This reinforces the association between large speed, the amount of shortening and force produced during shortening, and the magnitude of the FD to be partly dependent on the elongation vs shortening cycle to stop the phenomenon (6, 7, 32, 33).

One aspect that can be argued in studies of DF is about the practical effect of the phenomenon in daily life, given the limitation of studies using large muscle groups at submaximal and voluntary actions. This is evident when looking at the data in Table 1, which presents only three studies used voluntary stimulation. In this sense, one can say that there is a gap between the phenomenon itself and its applicability, which must be filled by new research to be undertaken.

Mechanisms and hypotheses for FD

The main hypotheses to explain the phenomenon of FD are inhibition-induced stress in the cross-bridges, the non-uniformity and instability of the sarcomere, the influx of H^+ ions and inorganic phosphate (PO_4^{3-}), and reducing the affinity between calcium (Ca^{2+}) and the active sites of actin, as detailed below.

Theory of stress-induced inhibition of cross bridges

This theory suggests a mechanical failure induced by stress generated in the active sites of actin - myosin coupling. This would occur in sites located in two helices of the F-actin chain, which suffer an angular distortion that produce rotation as the muscle is shortened at low speed. Thus, the active site of actin binding is displaced from its original position, preventing the head coupling of heavy mero-myosin or MCP chain (6, 7, 33, 43, 44).

Table 1 - Main characteristics of the studies on FD as regard to type of stimulation, material used and the observed effects considering the characteristics of the shortening applied

(To be continued)

Study	Material	Stimulation	Variable	Result
Abbot and Aubert (2)	Muscle	Eletric máx.	Speed (v) and range (e)	v↑ ⇒ FD↓ e↑ ⇒ FD↑
Edman et al. (14)	Fibers	Eletric submax.	Range	e↑⇒ FD↑
De Ruiter et al. (15)	Muscle	Electric sub. Voluntary	Force (F) by freq. of stimulation	F↑ ⇒ FD↑
De Ruiter and De Haan (16)	Muscle	Electric sub. Voluntary	Speed (v) e range (e)	v↑ ⇒ FD↓ e↑ ⇒ FD↑
McDaniel et al. (20)	Muscle	Electric sub.	Speed (v) e range (e)	v↑ ⇒ FD↓ e↑ ⇒ FD↑
Lee and Herzog (21)	Muscle	Electric max.	Speed (v) e range (e)	v↑ ⇒ FD↓ e↑ ⇒ FD↑
Rousanoglouet al. (24)	Muscle	Electric sub.	Speed (v) e range (e) in submaximal contraction (30% MVC)	v↑ ⇒ FD↓ e↑ ⇒ FD↑
Joumaa and Herzog (26)	Sarcomere	Electric max.	Imertion in Ca^{2+} (pCa = 3.5) and range (e)	e↑ ⇒ FD↑ even in Ca^{2+} rich solution (pCa = 3.5)

Table 1 - Main characteristics of the studies on FD as regard to type of stimulation, material used and the observed effects considering the characteristics of the shortening applied

(Conclusion)

Study	Material	Stimulation	Variable	Result
Lee et al. (27)	Muscle	Electric sub.	Speed (v), range (e)	v↑ ⇒ FD↓ e↑⇒ FD↑
McGowan et al. (31)	Muscle	Electric max. Voluntary	Speed (v), range (e), Stationary Ciclism at 90 rpm	v↑ ⇒ FD↓ e↑ ⇒ FD↑
Herzog and Leonard (33)	Fibers	Electric max.	Speed (v), range (e)	v↑ ⇒ FD↓ e↑ ⇒ FD↑
Herzog et al. (41)	Muscle	Electric max.	Speed (v), range (e)	v↑ ⇒ FD↓ e↑ ⇒ FD↑
Maréchal and Plaghki (43)	Muscle	Electric max.	Speed (v), range (e)	v↑ ⇒ FD↓ e↑ ⇒ FD↑

Note: MVC: maximal voluntary contraction; range: amount of shortening employed; pCa - Ca^{2+}: concentration.
Source: Research data.

The proponents of this hypothesis propose that an increase in the rate of cross-bridge uncoupling and an angular distortion, high enough to modify the organization, the structural arrangement and the ideal overlap between the actin-myosin quantity, alter the optimal sarcomere length. Such a change would be sufficient to induce a deformation in the active sites of actin-myosin. These then would substantially reduce the number of available cross-bridges, as well as the tension generated by each cross bridge (6, 7, 32, 33, 34, 43, 45, 46, 47, 48, 49).

Orlova and Egelman (50) preclude this hypothesis by proposing a memory of the double helix of F-actin chain, where the angular distortions would be very small. This memory would defeat the mechanism of inhibition induced by stress of cross bridges actin-myosin, which would result in the removal of the actin heads MCP active site.

Such memory would keep a kind of angular order to retain the position of the actin subunit in an appropriate coupling and formation of the cross-bridges (50) angle. On the other hand, for some advocates of FD (2, 6, 7, 19, 32, 33) subsidies to confirm it, use the analysis of the common features of the phenomenon. They take into consideration that it increases at large amplitudes of shortening, low speed and large amounts of force produced during active shortening.

Theory of non-uniformity and instability of sarcomere

The non-uniformity of sarcomeres theory is based on the fact that the sarcomeres have a mechanical property of non-uniformity and instability. This is because the sarcomeres (or half sarcomeres) do not reproduce the same rate of change in length imposed to the whole muscle tissue.

Thus, it is proposed that this non-uniformity would lead to depression of isometric force following an active pre shortening because while some sarcomeres would resume its original shape and length (observed during isometric contraction reference), others remain shortened even after the removal of stimulus (2, 6, 7, 14, 15, 16, 32, 34, 35, 36, 40, 51).

It should be said that, if one accepts this theory, it would be expected that the decrease in isometric force would have any relation to the LTC, since the maintenance of shortening at levels below the optimum length would be responsible for the reduction in force. That is what some proponents of this theory have reasoned, because the FD is a phenomenon observed primarily in the descending limb of LTC (6, 7, 32).

Furthermore, the FD should not occur in uniform sarcomeres, with constant and regular lengths before and after muscle shortening, as already noted (6, 7, 32). However, these arguments are refuted by studies

showing that the phenomenon also occurs, albeit to a lesser extent, in the ascending limb of the LTC.

Even in the presence of a control mechanism to stabilize and maintain uniformity, structural integrity and regulate sarcomere length, FD has been observed, also revealing a reduction in muscle passive tension (6, 7, 21, 32, 33, 34, 38, 39, 40).

In accordance with these recent studies, studies that investigated this hypothesis by manipulating sarcomere length, using stretch-shortening cycles, pre stretching or shortening only found conflicting results (7, 52, 53, 54). While indicating asymmetry, the results point to the stability and uniformity of sarcomeres and half sarcomeres, and ultimately call into question the validity and justification of the theory of non-uniformity and instability of sarcomeres.

Theory of reduced affinity between calcium ($Ca2^{+}$) and the active sites of actin

It has been attributed to Ca^{2+} a role of regulation and control in the active sites of actin, favoring the coupling mechanism and the actin-myosin cross-bridge activation. A decrease in affinity and sensitivity of active sites of actin to Ca^{2+}, the pumping rate of Ca^{2+} into the sarcoplasm toward the active actin sites, and the Ca^{2+} ATPase system activity has been linked to mechanisms of fatigue. Moreover, it has been pointed out as the probable cause of the mechanism of SCD and reduced muscle passive tension (55, 56, 57, 58, 59, 60, 61, 62).

It is also possible that the mechanism of inhibition by stress induced cross-bridge to actin-myosin responds not only to deformations and structural changes of the active actin-myosin coupling sites, but also to cellular Ca^{2+} dependent mechanism.

Although the Ca^{2+} dependent mechanism may encourage titin filaments, generating an increase in passive tension during contraction, it could induce a reduction in the rate of decoupling of the active sites of cross bridges (54, 63, 64, 65, 66, 67).

As opposed to the action of Ca^{2+} in the control of inhibition induced stress of cross bridges, it is suggested that two isoforms of troponin, troponin I (SSTN-I) and troponin C (sTn-C) exert greater influence on the dynamic coupling of actin -myosin filaments, especially thin filaments of actin, as compared to Ca^{2+} dependent (68, 69, 70) system. Moreover, taking into account the evidence presented, it can be thought that the mechanism of FD is only a transitory phenomenon, instantly abolished as soon as you recompose the sarcoplasmic Ca^{2+} concentrations in the muscle fiber (6, 7).

However, the FD has long duration, and cannot be completely abolished after the instantaneous removal of the stimulus of muscle shortening. This fact rule out the possibility of reducing the affinity of Ca^{2+} sensitivity as the determining mechanism for this phenomenon, or in other words, the phenomenon could be simply related to muscle fatigue (2, 6, 7, 15, 32, 33).

Theory influx of protons (H^{+}) ions and inorganic phosphate (PO_4^{3-})

Some researchers have argued that increases in the influx of H^{+}, and PO_4^{3-} into the muscle fiber are associated not only to the mechanism of muscle fatigue, but to the FD, decreased passive tension during the shortening phase. The muscle fiber size should also be taken into account, whereas the concentrations of H^{+}, and PO_4^{3-} in a proportional relationship that is, higher fiber size, the greater the influx of H^{+}, and PO_4^{3-} (40, 61, 71, 72, 73, 74, 75, 76).

As the both ions driven cannot be removed quickly, one can speculate that FD could not be abolished instantaneously after quick deactivation muscle, but should persist for a long period of time (6, 7, 32). However, although the phenomenon of FD present long term stimulus while there, it is abolished in relatively short periods of time, 0.5 s is 1, leading to the rejection of this theory to explain it (2, 6, 7, 32, 42).

On the other hand, it should be considered the arguments that support the long-term feature of the phenomenon (2, 6, 7, 33, 36, 40, 41). In addition, the contrast of the studies that show that relatively short periods that cause muscle deactivation may abolish instantly FD must also be taken into account (2, 6, 7, 32, 36, 51). Therefore, there is a need for future studies comparing the two hypotheses.

As can be seen, the phenomenon of FD is a fact that almost irrefutable, but its cause has conflicting rationales. It is important to reiterate that the phenomenon is not simply the reduction of isometric force after a concentric action due to muscle fatigue. It goes beyond that, even because the FE has many similarities with the FD and is associated with increased strength, even after maximal actions.

The concept of Force Enhancement

FE is defined as the increase in isometric force after active muscle stretching, when compared to a reference isometric contraction obtained in the same muscle length. In the same manner as in FD, the force produced has been measured in sarcomeres taken under stimulation, or muscle fiber. Few studies have been devoted to the investigation of the phenomenon in large muscles and submaximal voluntary actions. This can be observed with the summary presented in Table 2.

The main factors that influence the intensity of the increase in isometric force are the amplitude and speed of stretching (2, 6, 7, 33, 39). However the rate of stretching was not able to influence the amount of FE in isolated muscle fibers of animal specimens (17, 18, 77, 78).

Compared to FD, FE is characterized by having longer duration (greater than 25s), and also depends on the cycle of shortening, lengthening, or is reduced according to the amount of active stretching prior to shortening. Studies show that FE is instantly abolished if the amplitude of the preceding shortening is equal to or higher than the magnitude of stretching employed (36, 79).

As in FD, the occurrence of FE initially was linked only to the lower limb and the peak of the LTC (22, 23, 24). However, new findings indicate that FE is also observed with reasonable stability in the ascending limb of the LCT (39, 80, 81, 82). Similarly to FD, FE researches has employed the electrical and in most cases with maximum frequency (Table 2). However, although some authors justify their use in models of motor control and therapeutic procedures, it is still observed a large gap as its practical application (5, 7).

Table 2 - Main characteristics of the selected FE studies as on type of stimulation, the material used and the observed effects considering the characteristics of lengthening applied

(To be continued)

Study	Material	Stimulation	Variable	Result
Edman et al. (17)	Fibers	Electric max	Speed and limb of LTC	v↑ ⇒ FE more on DL
Edman et al. (18)	Muscle	Electric max	Speed and limb of LTC	v↑ ⇒ FE↑ more on DL
Lee and Herzog (22)	Muscle	Electric max and voluntary max.	Speed	v↑ ⇒ FE↑
Oskouei and Herzog (23)	Muscle	Electric max. and Voluntary submaximal	Speed and submaximal contraction	v↑ ⇒ FE↑ more on intensive contractions
Joumaa et al. (25)	Fibers	Electric max.	Range (e) and limb of LTC	e↑ ⇒ FE↑ on DL
Hahn et al. (28)	Muscle	Electric max. and voluntary	Range (e) and DL of LTC	e↑ ⇒ FE↑ active* and APF↑
Pinniger and Cresswell (29)	Muscle	Electric and voluntary submax.	Range (e)	e↑ ⇒ FE↑
Hahn et al. (30)	Muscle	Voluntary max e submax.	Range (e)	e↑ ⇒ FE↑
Herzog and Leonard (33)	Fibers	Electric max.	Speed and Range of shortening lengthening cycle	v↑ and cycle and a↑ ⇒ FE↑
Schachar et al. (37)	Muscle	Electric max.	Speed and DL of LTC.	v↑ ⇒ FE↑ on DL
Herzog and Leonard (39)	Muscle	Electric max. and submax.	Speed (v) and different parts of LTC and different freq. of Stimulation (f)	v↑ on SA and SD ⇒ FE↑ as higher f
Rassier et al. (65)	Fibers	Electric max.	Active range and passive (e) over sarcomeres and APF	e↑ ⇒ APF↑ on sarcomeres

Table 2 - Main characteristics of the selected FE studies as on type of stimulation, the material used and the observed effects considering the characteristics of lengthening applied

(Conclusion)

Study	Material	Stimulation	Variable	Result
Herzog and Leonard (67)	Muscle	Electric max.	Range (e) under APF and FE	e↑ ⇒ APF and FE↑
Edman and Tsuchiya (78)	Fibers	Electric max.	Passive tension (TP) and FE	TP↑ ⇒ FE↑
Herzog et al. (79)	Muscle	Electric max.	Speed (v) and range (e) on FE and on passive tension (TP)	v↑ or e↑ ⇒ FE and TP↑
Peterson et al. (80)	Fibers	Electric max.	Range (e) on AL of LTC	e↑ ⇒ FE↑
Lee and Herzog (85)	Fibers	Electric max.	Range (e) on plateau limb of LTC and comparison between FIR and FE	e↑ ⇒ FIR↑ and FE↑
Rassier and Herzog (88)	Fibers	Electric max.	Range (e) and coupling and decoupling kinetics of cross bridges (inhibitor action of 2.3-BDM)	e↑ and concentration of 2.3-BDM↑ ⇒ FE↑
Joumaa et al. (90)	Fibers	Electric max.	Titin and APF, and the Ca^{2+} dependence mechanism of titin	Ca^{2+}↑ ⇒ APF↑ explain (25%) of APF

Note: LTC: length x tension curve; FE: force enhancement; APF: FE due to increased passive force; RIF: residual isometric force; 2.3-BDM: butanodionemonoxime; AL: ascending limb of the LTC; DL: descending limb of LTC; *: not statistically significant.

Source: Research data.

Mechanisms and assumptions for FE

The main theories available to explain the phenomenon of FE are the theories of non- uniformity and sarcomere instability, and the recruitment of elastic components in parallel (CEP).

Theory of non-uniformity and sarcomere instability

This hypothesis is based on the principle described above, that the mechanics of deformation and alteration of sarcomere length does not follow a regular pattern, in which all units of sarcomeres are subjects to homogeneous deformation when a lengthening is applied to the sarcomere (6, 7, 17, 32, 35, 36, 51, 77, 83). It is suggested that the production of isometric force generated above the isometric contraction reference force would cause a small degree of deformation of some sarcomeres stretching while others suffer to a greater extent.

This would lead to great changes in length and reducing the overlap between the actin and myosin, resulting in fewer active cross-bridges. As a compensatory mechanism, sarcomere non-uniform and more fragile, when extended beyond the optimum length, recruit the CEP to restore the number of active cross-bridges in the strongest sarcomeres process known as sarcomeral popping (32, 82, 83).

On the other hand, some studies indicate that the sarcomeral popping (stretching, thinning and disruption) could result in a sarcomerogenesis mechanism with the addition of new sarcomeres from the division of fragile new sarcomeres and smaller units (32, 83, 84, 85).

To confirm this hypothesis, Lynn and Morgan (86) and Lynn et al. (87) showed the occurrence of sarcomerogenese after 5 days of training. Using a stretching protocol with progressive increases in amplitude until a point beyond optimum length, featured a sub-acute mechanism and acute adaptation, contrasting with the chronic process, which requires weeks or months (88).

Considering these arguments, some speculations on the theory emerged about FE: a) could not be observed in the ascending limb of the LTC muscle, b) should be instantly abolished in uniform sarcomeres, with constant and regular lengths, c) the magnitude of the increase in force would always be lower than the

amount of force obtained in isometric contraction of reference. These speculations are in agreement with the results obtained in some studies (2, 6, 7, 19, 32, 43).

Paradoxically, other studies have shown that FE also occurs, albeit to a lesser extent, in the ascending limb of the LTC in uniform sarcomeres. They also showed that the amount of FE may exceed the amount of isometric force measured during the performance of an isometric contraction of reference, in the same muscle length (6, 7, 18, 32, 81, 89).

Elastic components in parallel recruitment theory

This theory is based on the scanning mechanism of the elastic force in parallel, with the titin filament assuming the main role (32, 39, 66, 79, 80, 89, 90, 91, 92, 93). Such filaments would be responsible for producing the voltage required to maintain the centering, the length and the ideal overlap between the actin and myosin. Also, it keeps the active sites of actin in suitable for the coupling heads of MCP angular position, and ensure the maintenance of muscle passive tension due to its mechanical properties (94, 95, 96).

Labeit et al. (64) propose that the maintenance of muscle passive tension was still associated with the Ca^{2+} dependent mechanism, due to a high affinity and coupling capacity between Ca^{2+} and the active site of titin filaments. This possibly would play important role in stabilizing the muscle length during contraction while stretching.

Thus, the mechanism of FE was associated with a significant increase in isometric force generated by the passive force after an active stretch. Confirmed this hypothesis, the amount of muscle shortening employed to the muscle immediately before the stretching, if equal to or of greater magnitude (dose-dependent mechanism) probably should abolish completely the phenomenon by means of inhibition of the elastic passive components (6, 7, 32).

Theories based on the increase of tensile strength have been confirmed in part by studies that showed an association between the increase in isometric force after active stretching and increased passive force. This association is independent of the coupling of new cross bridges actin – myosin that remains after ceased the stimulus (7, 17, 22, 28, 33, 97).

As the foregoing shortening that precedes the elongation at magnitude equal to or greater than the elongation reduces to zero the FE, and nullifies the effect of the elastic force (passive), there is ample evidence that FE is caused by muscle elasticity characteristics are (7, 32, 33, 35, 79, 97).

Conclusion

The phenomena of FD and FE are well described in the literature and accepted among researchers being recognized as inherent properties, both in the sarcomere as the isolated muscle fibers and, to a lesser extent to the muscle groups. However, the explanation for these phenomena is not well established.

Based on this review, it is suggested that the theory of stress-induced inhibition of cross bridges to actin-myosin may be a plausible explanation for the FD. This is due to the fragility of the theories of sarcomere instability and non-uniformity of influx of H^+, PO_4^{3-}, and the decreased affinity of Ca^{2+}, as well as the active sites of actin filaments that do not themselves could explain the mechanism FD. After all, it should be remembered that all have been refuted by the available studies.

As for the AF phenomenon, there seems to be evidence that is due to a component of mechanical origin, related to the increasing number of cross-bridges from actin-myosin coupled in a strong state. This component is potentially influenced by the increased recruitment of the elastic components of passive force, assigning to this role titin filaments. The applicability of these phenomena, little is seen of practical study in everyday life situations.

However, carefully noting the results, one can corroborate the statement made by others, that it should be taken into account in models of motor control. There are strong indications that may play a role in therapeutic procedures.

Moreover, understanding the functional dynamics of physiological and mechanical properties that regulate the production of force can result in the discovery of new strategies for prescribing physical activity, or even potentiation of existing techniques employed in rehabilitation sciences. In this sense, it seems quite reasonable that further studies should be developed.

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