The Muscle Metaphor in Self-Regulation in the Light of Current Theorizing on Muscle Physiology

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Michael Richter and Joséphine Stanek

5.1 Introduction

In the last two decades, the strength model of self-regulation or self-control, respectively, (e.g., Baumeister 2002; Baumeister et al. 2000, 2007; see also Lopez et al. in press) has exerted a considerable impact on self-regulation research and theorizing. The model conceptualizes self-regulation as the capacity to alter one's own behavior (e.g., Baumeister 2002; Baumeister and Vohs 2007) and postulates that controlling behavior (for instance, overriding a habitual response) requires resources or energy, respectively. Resources that are mobilized to regulate behavior are supposed to be consumed in the regulation process and need to be restored. According to the strength model of self-regulation, all kinds of self-regulatory activity draw on the same resources. Performing a self-regulatory action should thus reduce the amount of resources that are available for subsequent self-regulation. Given that the model predicts that self-regulation efficiency is a direct function of the amount of available resources, performing a task that requires self-regulation should lead to decreased performance in subsequent tasks that rely on self-regulation. This effect is called ego-depletion effect and most of the empirical work on the strength model of self-regulation has focused on it (see Hagger et al. 2010, for a recent review of the empirical research on ego depletion).

When presenting the strength model of self-regulation, authors have repeatedly likened self-regulation to muscle activity arguing that self-regulation resembles a muscle—the muscle metaphor. The ego-depletion effect should resemble muscle fatigue—the decrease in muscle performance after sustained physical exercise that is restored after rest. Self-regulation and muscle activity should both require energy resources, and the depletion of these resources should underlie both the ego-depletion effect and muscle fatigue. The strength model of self-regulation also claims that self-regulation can be trained and strengthened like a muscle. Repeatedly performing self-regulatory tasks should lead to higher self-regulation capacities and better performance in tasks that require self-regulation.

In this chapter, we will take a closer look at the physiological foundation of the muscle metaphor. After an introduction to muscle functioning, we will discuss the two key elements of the muscle metaphor. We will elaborate on resource depletion as the cause of muscle fatigue, and we will discuss training effects on muscle strength.

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M. Richter (✉) · J. Stanek
Geneva Motivation Lab, FPSE Department of Psychology, University of Geneva, Bd. du Pont d'Arve 40, 1205 Geneva, Switzerland
e-mail: michael.richter@unige.ch

J. Stanek
e-mail: josephine.stanek@unige.ch

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We will show that the muscle metaphor relies on a simplistic concept of the muscle that does not adequately reflect the current state of research and theorizing in muscle physiology. We will also elaborate on how self-regulation research and theorizing may benefit from the physiological research on muscle work.

5.2 Muscle Work

5.2.1 Muscle Structure

Skeletal muscles contain bundles of parallel muscle cells, the muscle fibers.[1] Muscle fibers are mainly composed of cylindric myofibrils that extend the entire fiber length. Each myofibril is surrounded by a sarcoplasmic reticulum and consists of order arrangements of the proteins actin and myosin as well as other proteins that have a structural function or are involved in muscle action. The proteins form sarcomeres that lie in series and constitute the basic contractile unit of the muscle (see Fig. 5.1 for a schematic representation of muscle composition). Each sarcomere is limited by two sheets of structural proteins (Z proteins) running transversely across the fiber (Z line). Thin myofilaments composed of two helically coiled actin filaments and attached tropomyosin and troponin molecules project at right angles from the Z lines towards the sarcomere center. Thick myosin filaments lie in the center of the sarcomere paralleling the actin myofilaments. Actin and myosin filaments overlap but at rest the actin filaments do not reach the central region of the myosin filaments. Actin and myosin filaments build a hexagonal structure so that each myosin myofilament is surrounded by six actin filaments and each actin element is surrounded by three myosin filaments. The myosin molecules consist of a long tail and a globular head that projects towards the actin filaments. Both the myosin heads and the actin filaments contain binding sites for one another, but at rest the myosin–actin binding is prevented by tropomyosin and a troponin subunit (troponin-I) that block the binding site on actin (see Fig. 5.2).

5.2.2 Muscle Contraction

Muscles are innervated by alpha motor neurons originating from the central nervous system. Each alpha motor neuron innervates many muscle fibers. The electrical stimulation of the motor neuron causes an action potential at all innervated muscle fibers. The generated action potential propagates over the sarcolemma and enters into the muscle cell via a system of transverse tubules. The transverse tubules system is linked to the sarcoplasmic reticulum—the cell's store of calcium ions (Ca^{2+}). An action potential that is spread by the transverse tubules systems leads the sarcoplasmic reticulum to release the stored Ca^{2+} into the cytoplasm. The released Ca^{2+} binds to a troponin subunit (troponin-C) causing a structural change of tropomyosin that exposes the myosin-binding site on actin. Given that myosin–actin binding is no longer prevented, the myosin head attaches to actin and pivots. This "power stroke" causes a sliding of the actin filament towards the center of the sarcomere. Figure 5.3 displays this process.

In the default state—when myosin–actin binding is prevented by troponin-I—adenosine diphosphate (ADP) and inorganic phosphate (P_i) are bound to the myosin head. When the myosin head connects to actin—building a so-called cross-bridge—both are removed. After its power stroke, the myosin head stays connected to actin until the binding of adenosine triphosphate (ATP) to the myosin head causes another structural change that separates the myosin head from actin. The enzyme myosin ATPase then splits ATP to ADP and P_i that remain connected to the myosin head. The energy that is made available by this ATP hydrolysis is transferred to the myosin head causing the head to pivot back into its resting state. The sequence described above is called cross-bridge cycling and continues as long as Ca^{2+} binds to troponin-C—removing the blocking of myosin–actin binding—and as long

[1] Good general introductions to muscle physiology can be found in Brooks et al. (2005), McArdle et al. (2010), Scott (2008), Sahlin et al. (1998), Sherwood (2010), or Westerblad et al. (2010).

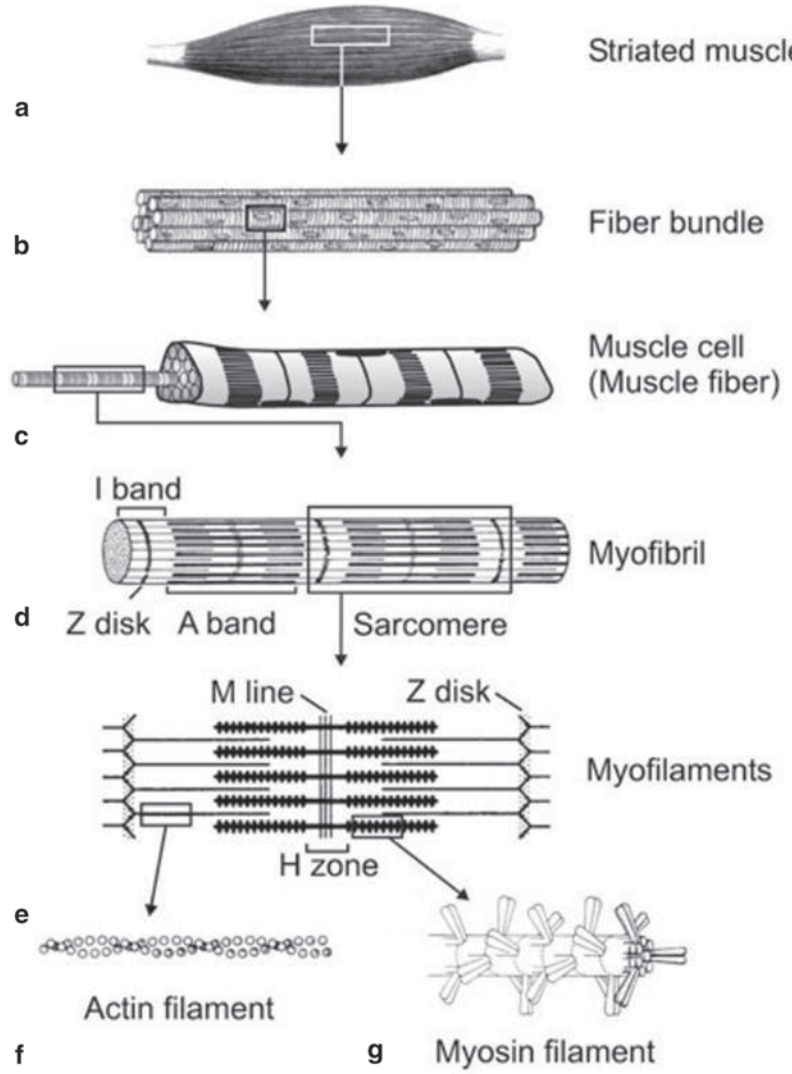

Fig. 5.1 Composition of skeletal muscle tissue. (From Functional anatomy of muscle: Muscle, nociceptors and afferent fibers by S. Mense, 2010, published in *Muscle pain: Understanding the mechanisms* by S. Mense and R.D. Gerwin, pp. 17–48. Copyright by Springer. Reprinted with permission)

as there is enough ATP to disconnect myosin and actin. The shortening of the sarcomeres caused by the repetitive cycling of myosin heads shortens the muscle fiber and creates force.

The membrane of the sarcoplasmic reticulum contains ATP-driven Ca^{2+} pumps that continuously pump Ca^{2+} from the cytoplasm into the sarcoplasmic reticulum. When the stimulation of the muscle ceases, that is, when there are no longer action potentials that lead the sarcoplasmic reticulum to release Ca^{2+} into the cytoplasm, cytoplasmic Ca^{2+} concentration quickly drops due to the activity of the pump. Due to the low cytoplasmic Ca^{2+} concentration, Ca^{2+} no longer binds to troponin-C and the tropomyosin–troponin complex changes back to its initial configuration blocking the myosin-binding site on actin. Given that myosin heads can no longer connect to actin, muscle contraction ends.

5.2.3 Energy Metabolism

ATP is the immediate energy source of muscle contraction; that is, it is the only energy source that muscle cells can directly use for contraction.

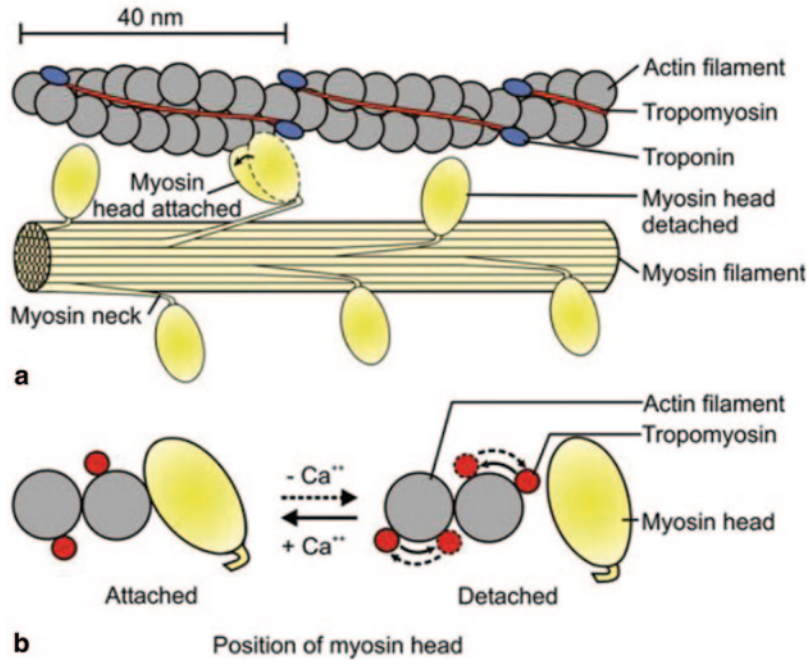

Fig. 5.2 Actin–myosin interaction. (From Functional anatomy of muscle: Muscle, nociceptors and afferent fibers by S. Mense, 2010, published in *Muscle pain: Understanding the mechanisms* by S. Mense and R. D. Gerwin, pp. 17–48. Copyright by Springer. Reprinted with permission)

Other energy-rich compounds (e.g., glucose, fatty acids) need to be broken down and their energy needs to be transferred to ATP before it is available to muscle cells. In skeletal muscle fibers, ATP is mainly used for cross-bridge cycling, pumping Ca^{2+} back into the sarcoplasmic reticulum, and the activity of the Na^{+}–K^{+} pump that restores the cell's resting membrane potential after an action potential (e.g., Homsher 1987; Kushmerick 1983). ATP is stored in muscle cells and releases energy when hydrolyzed by the enzyme adenosine triphosphatase (ATPase) to ADP and P_i ($ATP + H_2O \leftrightarrow ADP + P_i + H^+ + \text{energy} + \text{heat}$). Given that the amount of stored ATP is low—it would enable only a maximal contraction for one or two seconds—ATP has to be continuously restored. Three buffer systems—the phosphagen system, glycolysis, and oxidative phosphorylation—continuously resynthesize ATP to prevent ATP depletion. The buffer systems differ regarding the rate of ATP resynthesis and the total amount of ATP that they can produce. All three systems contribute to ATP resynthesis, but their relative importance varies with the duration and intensity of exercise (e.g., McArdle et al. 2010). At the onset of high-intensity exercise, most ATP comes from the phosphagen system, whereas glycolysis is the major source of ATP after approximately 30 s of exercise. After a minute of exercise, most ATP stems from oxidative phosphorylation.

5.2.3.1 The Phosphagen System

The phosphagen system constitutes the fastest way to resynthesize ATP (it may produce approximately 2.6 mmol s^{-1} kg^{-1} of ATP, Greenhaff et al. 2004). It comprises three main reactions. The creatine kinase reaction produces ATP by converting phosphocreatine (PCr), adenosine diphosphate (ADP), and a proton (H+) to ATP and creatine (Cr). The adenylate kinase reaction also resynthesizes ATP. It converts two ADP molecules to one ATP and one adenosine monophosphate (AMP) molecule. While the adenylate kinase reaction produces ATP, the amount of restored ATP is low compared to other sources of ATP resynthesis. The reaction plays a major role in removing ADP to keep the ATP/ADP ratio high, which is essential for ATP hydrolysis. Moreover, the produced AMP stimulates glycolysis by activating enzymes that are crucial for the rate of glycolysis. The third phosphagen reac-

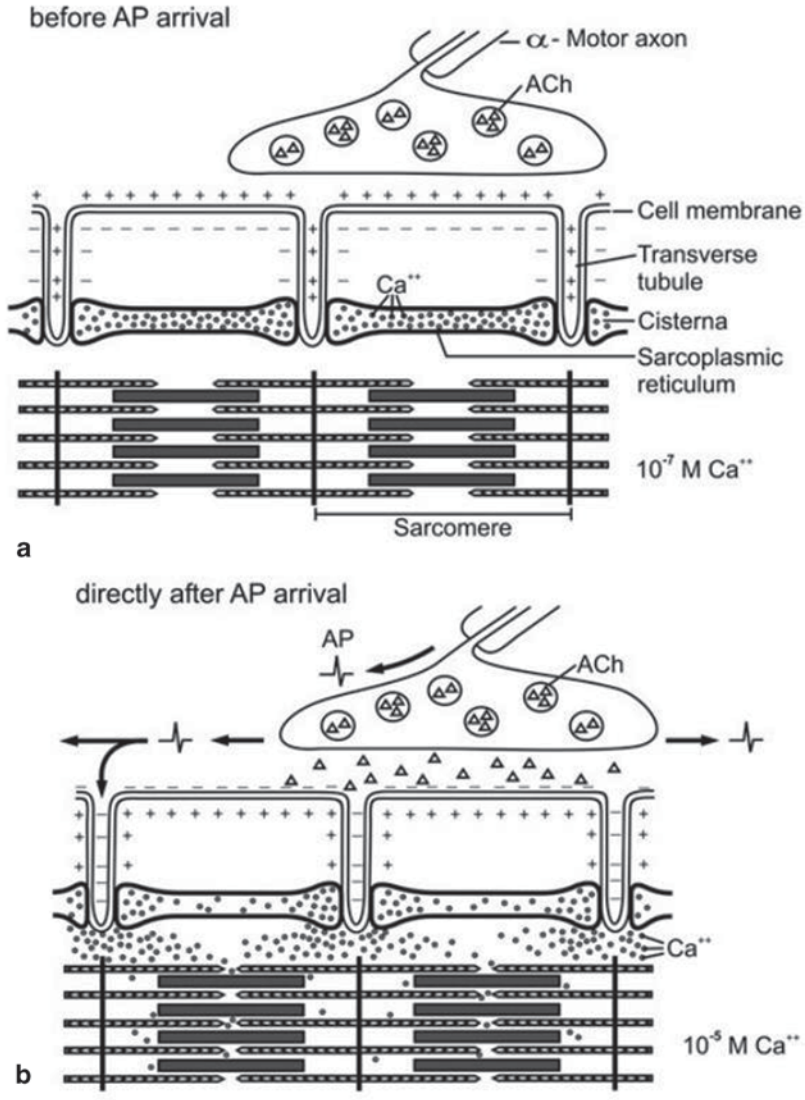

Fig. 5.3 Excitation–contraction coupling. (From Functional anatomy of muscle: Muscle, nociceptors and afferent fibers by S. Mense, 2010, published in *Muscle pain: Understanding the mechanisms* by S. Mense and R. D. Gerwin, pp. 17–48. Copyright by Springer. Reprinted with permission)

tion, the AMP deaminase reaction, does not produce ATP. It converts AMP and H_2O to ammonia (NH3) and inosine monophosphate (IMP). The AMP deaminase reaction removes AMP, which speeds up the adenylate kinase reaction and restores by this means a high ATP/ADP ratio. The phosphagen system—mainly the creatine kinase reaction—enables the muscle to rapidly regenerate a high amount of ATP. However, the amount of PCr stored in muscle fibers is relatively low and would only last for a contraction of a couple of seconds if it was the only source of ATP regeneration.

5.2.3.2 Anaerobic Glycolysis

Anaerobic glycolysis provides a second way to regenerate ATP. Its ATP turnover rate (approximately 1.4 mmol s^{-1} kg^{-1} of ATP, Greenhaff et al. 2004) is lower than that of the phosphagen system but higher than the turnover rate of oxidative phosphorylation. During glycolysis, blood glucose and muscle glycogen—glycogen is the stored form of glucose—are broken down to pyruvate yielding two molecules of ATP per molecule of glucose. Pyruvate is a substrate for oxidative phosphorylation. However, mitochondria need oxygen to produce ATP from pyruvate.

If pyruvate production exceeds the capacity of the mitochondria to take up pyruvate, pyruvate is transformed to lactate via the lactate dehydrogenase reaction (pyruvate + NADH + H+ ↔ lactate + NAD+). This prevents the accumulation of pyruvate, which would inhibit glycolysis. The production of lactate also helps glycolysis by regenerating NAD+, which is an essential substrate for glycolysis. Lactate production also delays metabolic acidosis—a potential cause of muscle fatigue—by contributing to metabolic proton buffering (e.g., Tiidus et al. 2012).

5.2.3.3 Oxidative Phosphorylation

Oxidative phosphorylation (synonyms are cellular oxidation, mitochondrial respiration) refers to the mitochondrial regeneration of ATP from macronutrients like carbohydrates, fat, or amino acids. Three major steps are involved in this process. First, the fuel molecules are broken down to acetyl-CoA. Second, acetyl-CoA is degraded in the citric acid cycle (synonyms are tricarboxylic acid cycle or Krebs cycle) in several steps. In this process, electrons in the form of hydrogen atoms are transferred from the substrates to coenzymes (nicotinamide adenine dinucleotide, NAD+, and flavin adenine dinucleotide, FAD) resulting in the generation of CO_2. Third, the electrons are transferred from the reduced coenzymes (NADH and $FADH_2$) to oxygen in the electron transport chain. This releases energy that is used to regenerate ATP from ADP and P_i.

In the oxidative phosphorylation of glucose (and glycogen), glucose is first broken down in anaerobic glycolysis to pyruvate that then enters the mitochondria where it is converted to acetyl-CoA. The complete oxidation of carbohydrates is relatively slow (e.g., approximately 0.68 mmol s^{-1} kg^{-1} of ATP for muscle glycogen, Greenhaff et al. 2004), but it is more efficient than anaerobic glycolysis (it produces 32 molecules of ATP per glucose molecule). The energy that the stored carbohydrates can provide (approximately 2000 kCal) exceeds the energy that is available from the phosphagen system. The largest source of potential energy (between 50.000 and 100.000 kCal) constitutes triaglycerols, stored fat. Most triaglycerol is stored in adipose tissue and can be hydrolyzed to yield one glycerol molecule and three fatty-acid molecules. Glycerol and fatty acids are transported in the blood to the muscle where glycerol serves as substrate for glycolysis to generate pyruvate that is converted to acetyl-CoA. Fatty acids are transformed in the mitochondria to acetyl-CoA in a process called beta-oxidation. Acetyl-CoA then enters the citric acid cycle to produce ATP as described above. The regeneration of ATP via oxidative phosphorylation of triaglycerol is very slow (0.24 mmol s^{-1} kg^{-1} of ATP for fatty acids, Greenhaff et al. 2004), but one molecule of triaglycerol may produce 460 molecules of ATP (19 ATP from glycerol breakdown, 441 ATP from the fatty-acid molecules). Amino acids only play a significant role in exercise exceeding 1 h (e.g., Poortmans 2004). In long-lasting exercise, they may provide up to 6% of the total expended energy. Amino acids contribute to ATP regeneration by serving as intermediates in the citric acid cycle or by being converted to pyruvate or acetyl-CoA. The specific metabolic pathways as well as the amount of regenerated ATP depend on the type of amino acid.

5.3 The Muscle Metaphor

5.3.1 Resource Depletion as a Cause of Muscle Fatigue

We will now take a closer look on how the muscle metaphor fits with the mechanisms described above. One key element of the muscle metaphor is that both muscle fatigue and decreased self-regulation efficiency are supposed to result from the depletion of energy resources. The muscle metaphor thus suggests that the decline in muscle performance after sustained exercise is due to a lack of energy. Given that ATP is the direct source of energy for muscle contraction, one might wonder if ATP depletion is the cause of muscle fatigue.

Empirical studies provided mixed evidence for this hypothesis. Some studies found no decrease in ATP concentration even if maximal muscle force was considerably reduced (e.g.,

Allen et al. 2002; Baker et al. 1993) or observed slight decreases in ATP that did not parallel the decrease in maximal muscle force (e.g., Baker et al. 1994; Dawson et al. 1978). Other studies provided positive evidence by demonstrating strong associations between decreased ATP availability and force decline (e.g., Karatzaferi et al. 2001), particularly, when looking at local ATP concentrations (e.g., Westerblad et al. 1998). It is of note that the reduced maximal force associated with decreased ATP concentration is not supposed to reflect a lack of energy available for muscle concentration. It has been attributed to decreased Ca^{2+} release from the sarcoplasmic reticulum resulting from reduced ATP concentrations (e.g., Allen et al. 2008; Westerblad et al. 1998). Given that the force-generating cycling of cross-bridges relies on the unblocking of myosin–actin binding sites by Ca^{2+}, a reduction in released Ca^{2+} results in less cross-bridge cycling and less force. The inhibiting effect of reductions in ATP on Ca^{2+} release seems to constitute a protection mechanism that reduces the energy consumed by cross-bridge cycling and reuptake of Ca^{2+} by the sarcoplasmic reticulum and prevents further decreases in ATP. Such a protection mechanism makes sense in the light of the serious consequences of complete ATP depletion. Given that ATP is the general energy source of the muscle cell, decreased ATP availability seriously impairs the cell's functioning, and leads to muscle rigor and cell death (e.g., MacIntosh et al. 2012; MacIntosh and Shahi 2011).

What about the energy systems that restore ATP? Do they show signs of depletion? One may speculate that these systems become depleted being no longer able to resynthesize ATP at a rate that is high enough to maintain a high-force level. As discussed above, the PCr system allows the muscle to restore ATP at a higher rate than the other systems. Thus, depletion of PCr stores could significantly slow down ATP resynthesis and the reduced ATP availability might lead to decreased force. The empirical evidence regarding the relationship between PCr depletion and force decline is mixed. Several studies showed that PCr concentration declines in parallel to declining maximal force at the onset of exercise and that PCr stores can become completely depleted (e.g., Sahlin et al. 1987). However, other studies cast doubt on the role of PCr depletion as a causal agent of muscle fatigue. They showed that PCr and maximal force during exercise and recovery can be dissociated (Fitts and Holloszy 1976; Saugen et al. 1997) and that PCr stores can still be very high even when participants are completely exhausted (e.g., Sahlin et al. 1992).

There is some evidence for associations between falling muscle glycogen and blood glucose concentrations and fatigue during long-lasting, high-intensity exercise (e.g., Callow et al. 1986; Chin and Allen 1997). However, there are other studies showing that this relationship only holds for a certain range of exercise intensity. If exercise intensity is low or very high, glycogen concentration and fatigue are dissociated (e.g., Saltin and Karlsson 1971). Furthermore, there is also evidence suggesting that reductions in muscle glycogen do not automatically lead to reduced ATP resynthesis (Baldwin et al. 2003) challenging the hypothesis that a lack of energy underlies the observed relationship between reductions in glycogen concentration and muscle fatigue. Depletion of triaglycerol, stored fat, has not been discussed as a major cause of fatigue. This is not surprising given that the amount of triaglycerol stored in adipose tissue provides enough energy to run more than 30 marathons.

To sum up, there is some evidence that the availability of ATP and of substances that are used to restore ATP can be reduced after sustained physical exercise and that these changes may parallel muscle fatigue. However, there is also evidence that questions the hypothesis that energy depletion is a major cause of fatigue and evidence for complete depletion of energy resources is sparse. The depletion of energy resources is only one among several factors that have been discussed in the physiological literature as a cause of muscle fatigue (see Allen et al. 2008; Fitts 1994; Gandevia 2001; Sahlin et al. 1998; Westerblad et al. 1998, 2010, for reviews). Muscle contraction involves several steps and an impairment of any of these steps may lead to a decreased muscle performance. Muscle fatigue resulting from events localized in the cerebral cortex (e.g.,

impaired descending output to motor neurons) or in the spinal cord (impaired alpha motor neuron firing, suboptimal recruitment) is called central fatigue. Peripheral fatigue refers to impairments that take place in the muscle itself. Factors causing peripheral fatigue include metabolic inhibition of cross-bridge cycling, impairment of excitation–contraction coupling, and impaired neuromuscular transmission. Metabolic factors that have been extensively discussed in the physiological literature as significant causes of muscle fatigue are the accumulation of inorganic phosphate (P_i), lactate, and hydrogen ions (H^+).

P_i is the product of the breakdown of PCr. High levels of P_i seriously impair muscle functioning by reducing the force produced by cross-bridges, decreasing myofibrillar sensitivity to Ca^{2+}, and reducing the amount of Ca^{2+} released by the sarcoplasmic reticulum (e.g., Allen et al. 2008; Westerblad et al. 2002, for reviews). The major causal role of P_i in muscle fatigue is further supported by the close relationship between P_i concentration and muscle fatigue during exercise and recovery (e.g., Fitts 1994). The accumulation of lactate produced by anaerobic glycolysis has also been suggested as a major cause of muscle fatigue. However, recent research calls this hypothesis into question (e.g., Allen et al. 2008) and recent reviews of muscle fatigue do not consider lactate accumulation to be a major cause of fatigue. Anaerobic glycolysis also increases H^+ concentration and decreases muscle pH. Increases in H^+ inhibit the enzyme phosphofructokinase reducing the rate of glycolysis, decrease myofibrillar Ca^{2+} sensitivity, reduce ATPase activity, impair cross-bridge functioning, slow down maximal shortening velocity of muscle fibers, and inhibit Ca^{2+} uptake and subsequent Ca^{2+} release by the sarcoplasmic reticulum (e.g., Fitts 1994, 2004; Westerblad et al. 2010, for reviews). All these factors considerably impair muscle functioning and lead to decreased maximal force. However, researchers recently started to question the importance of the slight reductions in pH observed in fatigued muscles suggesting that accumulation of H+ plays a minor role for human muscle fatigue (e.g., Westerblad et al. 2002, 2010).

In the preceding paragraphs, we have presented some of the factors and mechanisms that have been discussed in the physiological literature as causal agents of muscle fatigue. Despite decades of research on muscle fatigue, physiologists do not agree regarding the factors that cause fatigue and the relative importance of these factors (Fitts 1994). It is obvious that the muscle metaphor does not adequately reflect the physiological work on muscle fatigue and the controversy among researchers. By suggesting that there is agreement that energy depletion is the cause of muscle fatigue, the muscle metaphor overstates the role of depletion, overstates the degree of agreement among muscle physiologists, and neglects the variety of factors that seem to play a role in muscle fatigue. Depletion of energy resources may be a factor involved in muscle fatigue but it is neither the sole nor the major cause of muscle fatigue. Moreover, it is important to note that the muscle metaphor's depletion hypothesis conflicts with current ideas that many metabolic changes that cause muscle fatigue constitute protection mechanisms that prevent depletion (e.g., MacIntosh & Shahi 2011, 2012). Muscle fatigue may not be due to resource depletion but protect against it.

5.3.2 Training Effects on Muscle Strength

The muscle metaphor also suggests that repeatedly performing self-regulatory activity leads to increases in self-regulation efficacy like a muscle becomes stronger with repeated muscle exercise (e.g., Baumeister 2012; Baumeister et al. 2006, 2007). The muscle metaphor thus implies that repeatedly exercising a muscle leads to increased muscle strength. We will provide two examples that demonstrate that this constitutes a strong simplification of the complex relationship between muscle exercise and muscle adaption. First, muscle-training effects depend on the principle of overloading (McArdle et al. 2010). Only if the muscle is stimulated by repeated exercise at an intensity level that is higher than the normal exercise intensity level, adaptions will occur. Simply stimulating the muscle at a low intensity

will not result in any significant changes. Second, the specific effects of training depend on the type of exercise as well as on the intensity, frequency, and duration of the exercise (e.g., Brooks et al. 2005; McArdle et al. 2010). Not all types of muscle training lead to increases in muscle strength.

Training types may be classified into endurance and resistance training. Endurance training is characterized by a high-activation frequency of motor units and a modest load that the muscle contracts against (e.g., cycling, jogging). The specific effects of endurance training depend largely on the energy systems that are used during training. Training of the aerobic system increases, among others, mitochondria number and size, the number of enzymes involved in oxidative phosphorylation, the muscle's capacity to oxidize fatty acids, the capacity to oxidize carbohydrates during maximal exercise, and the size of slow-twitch muscles (e.g., Brooks et al. 2005; Holloszy and Coyle 1984; McArdle et al. 2010). Training of the anaerobic system increases anaerobic substrate (ATP, PCr, muscle glycogen) levels, the amount and activity of the enzymes that control anaerobic glycolysis, and the capacity to tolerate higher blood–lactate levels (e.g., Brooks et al. 2005; McArdle et al. 2010). Endurance training also leads to an increase in maximum blood flow to the activated motor units as well as an increased performance of the capillary system. The adaptions induced by endurance training basically allow the muscle to perform longer.

Resistance training refers to exercise that is characterized by a high load that the muscle has to contract against (e.g., weight lifting). It results among others in an increase in the cross-sectional area of the muscle (mainly due to increases in the cross-sectional area of individual muscle fibers), an improved capacity for motor unit recruitment, and an increased motor neuron firing efficiency (e.g., Fry 2004; Kraemer et al. 1996). The main effect of resistance training on muscle performance is an increase in maximal muscle force.

As is evident from this brief review, muscle training is a complex topic and training a muscle may lead to different outcomes depending on the type of training. The muscle metaphor's hypothesis that muscle strength increases as a function of repeated exercise inadequately represents the complex relationship between different types of muscle training and various effects on muscle performance.

5.3.3 The Utility of the Muscle Metaphor

In the preceding sections, we have sketched the mechanisms that underlie muscle work, muscle fatigue, and training effects on muscle performance. It should have become evident that muscle activity is a highly complex process and that there is still a controversy regarding the involved mechanisms. The muscle metaphor that is used in the literature on the strength model of self-regulation does not adequately reflect this complexity and controversy of current theorizing on muscle functioning. It relies on a simplistic idea of the muscle that conflicts in part with the physiological evidence.

The crucial question is whether the simplified model of muscle functioning that is implied by the muscle metaphor constitutes a problem. One reason for using a metaphor is to provide an analogy that facilitates the comprehension of a new subject by transferring information from a known subject to the new subject (Boyd 1993). The muscle metaphor could help people that are not familiar with the strength model of self-regulation to understand the model. Imagine a physiologist who is an expert in muscle physiology but knows nothing about the strength model of self-regulation. When she learns that self-regulation resembles a muscle, she will transfer the knowledge that she has about muscle physiology to self-regulation. She probably would expect that theorizing about self-regulation involves ideas about different energy systems that restore a primary energy compound (like the PCr system, anaerobic glycolysis, and oxidative phosphorylation restore ATP), a detailed model on how self-regulation relates to task performance (like the physiological model of muscle activation and contraction), a whole bunch of variables that are discussed as causes of self-regulatory failure (like the variety of variables that are discussed as causes of

muscle fatigue), and predictions regarding the specific effects of different self-regulation trainings on various performance parameters (like the specific effects of different types of muscle trainings). When she then learns about the predictions of the strength model of self-regulation, she will be confused by the fact that the model does not refer to the elements that she was expecting. It is obvious that her prior knowledge about muscle physiology would not help her to understand the strength model of self-regulation.

What about a person who is not familiar with the literature on muscle functioning? The muscle metaphor would not be of any help because this person could not apply the metaphor. Given that she has no prior knowledge about the muscle that could be transferred and facilitate the understanding of the strength model of self-regulation, the muscle metaphor would be useless. However, one might argue that everyone has some knowledge about muscle functioning that could be used. It is likely that everyone has gone through the experience that after intense physical exercise, muscle performance declines. People could draw on this knowledge to benefit from the muscle metaphor. However, even in this case the muscle metaphor relies on an oversimplification. Muscle fatigue can only be observed when maximal performance is assessed. If one does not have to exert one's maximum force, one will not observe fatigue effects (e.g., Allen et al. 2008). If one only has to perform at a submaximal level, one will be able to perform for a long time without any signs of fatigue. Imagine that you and your friend are asked to run 1 km. You have to run at your maximum speed, whereas your friend has to walk the kilometer at a speed of 1 km/h. Directly after the first task, both of you have to walk 2 km at a speed of 2 km/h. Even though you will be certainly more exhausted than your friend after the first task, there will be no performance differences between both of you in the second task. Both of you will easily manage to walk the 2 km in 1 h. The muscle metaphor neglects that muscle fatigue effects require the exertion of maximal performance in the second task. It simply suggests that performing high-intensity muscle exercise reduces performance in a second task, independent of the intensity of muscle activity that is required in the second task. Thus, people may have some personal experience regarding muscle functioning that may help them to understand some of the predictions of the strength model of self-regulation. However, their personal experience may also contradict the model's predictions—like in the example presented above—and hamper the understanding of the model.

It seems that the muscle metaphor's utility for facilitating the understanding of the strength model of self-regulation is limited. Unfortunately, using the muscle metaphor in self-regulation research leads to two serious problems. First, given that it does not adequately reflect current physiological thinking about muscle functioning, self-regulation researchers who present the muscle metaphor in their own scientific work are at risk of misrepresenting physiological knowledge. We think that scientists should aim at presenting knowledge from other domains as precisely and correctly as possible and avoid unwarranted simplifications. There is hardly a good reason why self-regulation researchers should inadequately present physiological knowledge in their own research. Second, we are afraid of the impact that the muscle metaphor will have on self-regulation researchers' thinking about the muscle. Individuals who do not have much knowledge about muscle physiology—and most self-regulation researchers probably do not know much about this topic—will adopt the idea that self-regulation resembles a muscle and will infer from the strength model's predictions to muscle work. For instance, they will adopt the idea that muscle fatigue is due to resource depletion and probably repeat this incorrect statement in their own work. Thus, the muscle metaphor may lead self-regulation researchers to develop incorrect representations of muscle functioning. Given the limited utility of the muscle metaphor and the discussed problems, we are wondering if the strength model on self-regulation would not be better off without the muscle metaphor. We are convinced that the model itself is strong enough to get along without the inadequate muscle analogy.

5.4 What Self-Regulation Researchers Can Learn from the Physiological Research on Muscle Functioning

Even if we think that the muscle metaphor is inadequate, we nevertheless feel confident that self-regulation researchers may benefit from the work on muscle physiology. The physiological research may provide a useful model that demonstrates how focused research on the mechanisms that underlie an effect may lead step by step to a deeper understanding of the effect. It also highlights that it is not the effect itself that merits scientific inquiry but the detailed understanding of the mechanisms that underlie the effect. Physiologists do not conduct extensive research on the effect that sustained muscle exercise results in decreased maximal muscle force. They are more interested in examining the causal mechanisms that underlie this phenomenon developing more and more sophisticated models of muscle fatigue. Self-regulation researchers still seem to be more interested in demonstrating ego-depletion effects instead of examining in detail the mechanisms that underlie the effect. In the light of more than 100 studies that have demonstrated ego-depletion effects—Hagger et al. (2010) already included 83 studies in their meta-analysis—this focus on replicating ego-depletion effects does not seem to be warranted. Furthermore, we doubt that such a focus fosters our understanding of ego-depletion effects and self-regulation.

There are nevertheless exceptions. A couple of researchers have discussed and examined mechanisms that should underlie the ego-depletion effect. For instance, Baumeister and colleagues suggested resource depletion as underlying mechanism (e.g., Baumeister 2002; Baumeister et al. 2000). Gailliot et al. (2007) postulated that glucose would be the resource that becomes depleted after self-regulatory efforts. Baumeister and colleagues recently speculated that the strategic conservation of resources may also play an important role (e.g., Baumeister 2012; Baumeister et al. 2007). A similar view was expressed by Beedie and Lane in their resource-allocation model of self-control (Beedie and Lane 2012). Further examples constitute Molden and colleagues' suggestion that ego-depletion effects are due to a lack of motivation (Molden et al. 2012), Job and colleagues' idea that subjective beliefs about the availability of resources are crucial (Job et al. 2010), Kaplan and Berman's attention restoration model that postulates that the depletion of voluntary, directed attention causes the ego-depletion effect, Tops and colleagues hypothesis that ego depletion reflects the protective inhibition of self-regulation and motivation (see Chap. 6), Inzlicht and Schmeichel's proposition that shifts in motivation and attention cause the ego-depletion effect (Inzlicht and Schmeichel 2012), or Wright's research that suggests that at least some ego-depletion effects might be due to feelings of fatigue (e.g., Wright et al. 2013).

These developments are steps in the right direction but, compared to theorizing on muscle functioning, current theorizing on ego depletion is still not very sophisticated and lacks important details. For instance, many central concepts like self-regulation, resources, or motivation are not well defined, which prevents from crucial empirical tests. Current models also do not include testable predictions on how self-regulation impacts performance or on how resources, glucose, motivation, attention, or subjective beliefs are translated into performance.

We agree with Inzlicht and Schmeichel (2012) that it is time to stop replicating one and the same effect and to start exploring the mechanisms that underlie the ego-depletion effect. The research on self-regulation would greatly benefit from empirical research and theorizing that focuses on the "black box" between "high self-regulatory effort in task 1" and "reduced performance in self-regulation in task 2." Researchers should aim at building models that define the central variables in a specific manner, provide detailed mechanisms that explain how self-regulation affects task performance, and postulate testable mechanisms that explain why and under which conditions exerting self-regulation in one task leads to decreased performance in subsequent tasks. Given that the central outcome that all models try to explain is task performance, every model that does not include specific predictions regarding

the determinants of task performance would be incomplete. The physiological research on muscle activity may provide a guiding model for the development of these models. Knowledge about current physiological research and theorizing on muscle work would at least prevent researchers from using muscle analogies that may seem to be convincing at first sight but that inadequately reflect current theorizing on muscle functioning.

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