Interdisciplinary Description of Complex Systems 2(1), 1-11, 2004

A KINETIC MODEL OF THE MUSCULAR CONTRACTION

B. Gaveau[1], M. Moreau[2, *] and B. Schuman[2]

[1]Laboratoire Analyse et Physique Mathématique,
Paris, France

[2]Laboratoire de Physique Théorique des Liquides,
Paris, France

SUMMARY

In this article, we build a self consistent mean field deterministic model for the muscular contraction. The two main variables are the number of free myosin heads and the number of myosin heads attached to the actin, just after attachment. The model is natural in the sense that it respects the physico-chemical natural constraints. We calculate the stationary state, prove that it is stable and calculate the efficiency.

KEY WORDS

muscular contraction, chemical kinetics, efficiency

CLASSIFICATION

PACS: 82.20.-w, 87.10.+e, 87.19.Ff

*Corresponding author, η: moreau@lptl.jussieu.fr; +33 1 4427 5947;
T. 16-15, 5ème étage, case 121, 4, Place Jussieu, 75252 Paris Cedex 05

INTRODUCTION

The metabolic process of a cell transforms chemical energy stored in bonds of various nutrients, either into other form of chemical energy, or into various forms of mechanical energy for transport of metabolites inside a cell, or communication between cells. Many forms of transport take place through membranes, or along special structures inside the cytoplasm. The muscular contraction is of the last category: it transforms the chemical energy stored in the phosphate bond of ATP in mechanical energy of the motion of a large protein called actin. The general character of this transformation is now well understood, at least in a qualitative manner, and is described in many text books (see [1, 2]). The first mathematical treatment of this process was given by Hill (see [3], for the first model, as well as [4], [5] and Hill's textbook for a more general theory of the conversion of chemical free energy into other forms of free energy [6]). More recently, there has been a renewed interest for mathematical models of molecular motors, as they are now called. These models follow the fundamental idea of Hill, namely that a certain degree of freedom can transit between two different form of free energy through a chemical reaction (see [7] for a recent review and references, as well as [8], [9], and also [10] for other type of mesoscopic motors like thermal ratchets). Nevertheless, certain of these models present difficulties of a mathematical nature, or difficulties concerning biochemical rate constants or of statistical nature. In a previous publication [11], we have introduced a detailed microscopic model, taking into account the actual fluctuations of the number of myosin heads which are attached to the actin filament. We have studied, in particular, the correlation between the number of heads which are attached to the actin filament and the actual force which is exerted on the actin molecule.

In this article, we define a new and simple model: this model is a self-consistent mean field theory of the muscular contraction. It can be described by three processes, two of them being of chemical nature, one of them being of a mechanical nature describing the motion of the actin. The total work and the efficiency can be calculated exactly. This model describes a different regime from [11], namely the case of an intermediate regime where the muscle is loaded and starts to contract, but not enough so that the spring reaction is important. It is much simpler than our previous model [11], but it also takes into account, although schematically, the chemical reactions of attachment and detachment of a myosin head in a plausible way.

The contents of this article are as follows: in section 2, we recall the biochemical description of the muscular contraction, in particular, we distinguish four phases. Only one of them will be the subject of this article. In section 3, we describe the self consistent kinetic model. We prove that it is natural, in the sense that it respects the natural physico-chemical constraints, calculate its stationary state and prove that it is stable (see section 4). We compute the work and the efficiency in section 5 and finally we discuss the various assumptions and intrinsic limitations of our model in the conclusion.

DESCRIPTION OF THE MUSCULAR CONTRACTION

A muscular cell is a specialized cell which expresses certain types of proteins which are able to transform chemical free energy into mechanical energy. The two essential proteins species which allow this transformation are a large rigid protein called actin and a shorter one called myosin which contains two articulate parts. The actin is attached to the wall of a sarcomer which is a compartment of the muscular cell. The

moving arm of the myosin can attach to the actin and then exerts a dragging force on the actin inducing the contraction of the sarcomer. The actin molecules and the fixed parts of the myosin molecules are arranged in separate parallel bundles. The moving arms of the myosin molecules spring out of the bundles of myosin. The arms are constituted of a linear peptidic chain surrounded by a global protein called the head of the myosin, which can attach to the actin. Many myosin heads can attach to a given bundle of actins (see Fig. 1 and ref. [1, 2]).

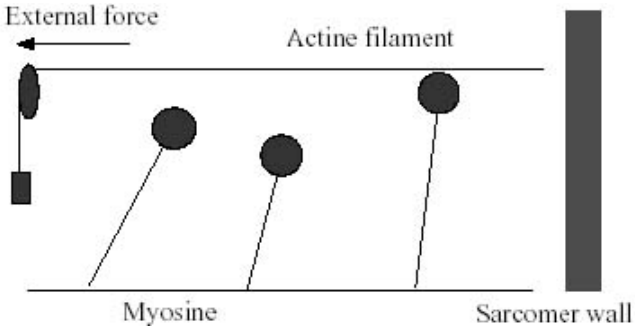

Figure 1. The interaction between the actine and the myosine heads molecules.

The myosin head has also a catalytic function for the hydrolysis of ATP (adenosine triphosphate) in ADP (adenosine diphosphate) + P (phosphate) with the release of an important amount of energy when the phosphate bond is broken (at normal temperature and neutral pH, the free energy released is about 30 kJ/mol). The molecule ATP and its phosphate bond are the essentially universal storage of energy which is used in the metabolism of all living organisms (see ref. [1, 2]).

We describe now the various states and phases of the contraction of a muscular cell.

1st phase. The unloaded cell at rest.

At rest, the myosin heads cannot attach to the actin molecules. Indeed, there is another protein called the troponine which blocks the sites of attachments on the actin, preventing the attachment of the myosin heads. The myosin arms can rotate and when the corresponding head is not attached, the angle θ between the fixed part of the myosin and the moving part takes its equilibrium value which is about $\pi/2$. Moreover, in this phase, the myosin head carries on a special site a dissociated ADP+P.

2nd phase. The effect of a nervous impulse.

The excitation of a muscle cell by a nervous impulse induces a release of the neurotransmittor acetylcholin, which allows ions Ca^{2+} to enter the cell, diffuse and inhibit the troponine which was blocking the attachment sites of the actin molecules for the myosin heads. The details of this process do not concern us here. But as a consequence, the myosin heads can now attach to an attaching site of the actin molecules.

3rd phase. The cycle of the myosin head (see Fig. 2).

Let us consider now a free myosin head equipped with its ADP+P which is in its equilibrium position, the angle between the fixed part of the myosin and the arm being $\pi/2$. This is step 1.

The myosin head together with its ADP+P attaches to a site of the actin forming a strong covalent bond, while the ADP+P detaches from the complex (step 2). Now, the

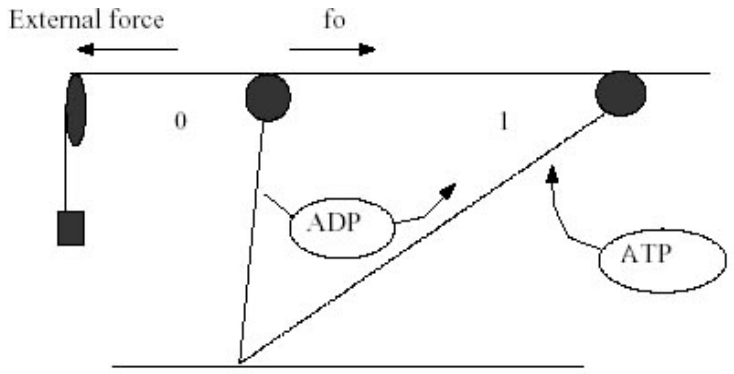

Figure 2. A myosine head has attached in position 0 to the actine and exerts a dragging force f_0. The ADP leaves the myosine head, and the myosine head reaches its new equilibrium position 1. An ATP attaches to the myosine which is then detached from the actine.

arm of the myosin is no more in its equilibrium position. The potential acting on the angle θ is such that the new equilibrium position of this angle, when the myosin head is attached to the actin, is $\pi/6$. So the myosin arm starts to rotate to reach this new equilibrium value. But as it is rigidly attached to the actin, it drags the actin inducing a contraction of the cell (step 3). During this motion of the myosin head coupled to the actin, an ATP molecule can attach to the myosin head and destabilize the bond between the actin and myosin head so that the myosin head is detached from the actin (step 4) and comes back to its free equilibrium position, while catalyzing the hydrolysis of the ATP molecule in ADP+P coming back to step 1. The myosin head equipped with the ADP+P molecules is ready for another cycle.

In each cycle, the actin moves by about 10 nm. One molecule of ATP is consumed, one molecule of ADP and a phosphate residue P are released.

4th phase. Coming back at rest.

Finally, this cyclic motion of the myosin head stops when the neural firing stops: the neurotransmitter is no more released, the influx of the ions Ca^{2+} stops, and the troponine which was inhibited, is disinhibited and blocks again the site attachment of the myosin head on the actin filaments.

In this article, we are interested in the third phase, when the attachment sites of the myosin heads on actin filaments are not blocked. On a given actin filament, there are many attachment sites (of the order of 10^3) and a corresponding number of myosin heads can reach the attachment sites, and when attached, can drag the actin filaments.

The cyclic motion of a myosin head is converted in the linear motion of the actin filament (and the contraction of the muscle), exactly as in a standard engine, where the cyclic motion of the piston is converted to a linear motion.

The main difference with a standard engine is that there are many "pistons" dragging the actin filament and causing the mechanical motion.

We can consider the attachment times of the various myosin heads, as well as the detachment times of the myosin heads as independent random variables, essentially exponentially distributed. On the other hand, the myosin heads which are attached to the actin filament have, while they stay attached, the same velocity, which is the velocity of the center of mass of the actin filament.

Following the analysis of Hill [1, 6], we assume that the potential energy of the angle θ between the fixed part of the myosin and the moving arm is

1. a given function $V^{(0)}(\theta)$ with a minimum at $\theta^{(0)} \approx \pi/2$ when the myosin head is not attached to the actin.
2. another given function $V^{(1)}(\theta)$ with a minimum at $\theta^{(1)} \approx \pi/4$, when the myosin head is attached to the actin (see Fig. 3).

The potential energy $V^{(1)}(\theta)$ is the potential energy of the angle θ of an attached myosin head, if it was alone. Its derivative is essentially the force exerted by the attached myosin head on the actin, if that myosin head were alone.

If we assume that all the free energy of the phosphate bond is released in mechanical energy, the force that a myosin head exerts on an actin filament is about

$$F \approx \Delta G/d \approx 0{,}5\cdot 10^{-19}/10^{-8} \approx 0{,}5\cdot 10^{-11} \text{ N/molecule.}$$

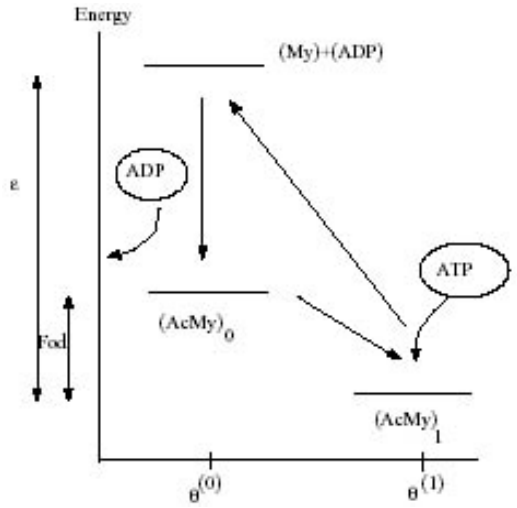

Figure 3. The cycle of the myosine head. This displays also the levels of energy with $\varepsilon > f_0 d$.

A SELF-CONSISTENT MODEL FOR THE ACTIN MOTION

We denote by M the total number of myosin heads. We simplify the description by assuming that the given myosin head can be in three different states (see Fig.3).

(i) unattached myosin heads, the number of which is denoted [My]

(ii) attached myosin heads with an angle θ close to the angle $\theta^{(0)}$; The number of these heads is denoted $[\text{AcMy}]_0$. When a head is in this state, it exerts a dragging force f_0 on the actin filament.

(iii) attached myosin heads with an angle θ close to the equilibrium value $\theta^{(1)}$. The number of these heads is $[\text{AcMy}]_1$. When the head is in this state, it exerts dragging force f_1 on the actin filament.

We assume that, when the myosin head is detached, it relaxes to equilibrium instantly in the potential $V^{(0)}$.

Obviously, one has the conservation law

$$[\text{My}]+[\text{AcMy}]_0+[\text{AcMy}]_1 = M. \quad (1)$$

One can describe the cyclic motion of the myosin head using three types of transitions.

(i) a reaction of attachment of the myosin head (with the ADP+P_i complex to the actin at an angle close to $\theta^{(0)}$, and the corresponding reaction of detachment of the myosin head which has received an ATP. These reactions are written

$$(\mathrm{My}) \underset{\bar{k}_1}{\overset{k_0}{\rightleftharpoons}} (\mathrm{AcMy})_0. \tag{2}$$

Notice that $\bar{k}_0$ is essentially proportional to the concentration of ATP and we assume that $\bar{k}_0 << k_0$.

(ii) a reaction of detachment of the myosin head which is attached to the actin at an angle close to $\theta^{(1)}$ (the myosin head has just received an ATP molecule) and a corresponding reaction of attachment of the free myosin head to the actin at an angle close to $\theta^{(1)}$

$$(\mathrm{AcMy})_1 \underset{\bar{k}_1}{\overset{k_1}{\rightleftharpoons}} (\mathrm{My}). \tag{3}$$

Notice here that k_1 is essentially proportional to the concentration of ATP and that $\bar{k}_1 << k_1$.

The notations are chosen so that $\bar{k}_j << k_j, j = 0, 1$.

(iii) a mechanical transition, when the attached myosin heads transits from an angle close to $\theta^{(0)}$ to an angle close to equilibrium $\theta^{(1)}$ or vice versa

$$(\mathrm{AcMy})_0 \underset{\omega_{01}}{\overset{\omega_{10}}{\rightleftharpoons}} (\mathrm{AcMy})_1. \tag{4}$$

Now, each attached myosin head has the same velocity v which is the velocity of the center of mass of the actin filament. So, the rates of the transitions in (4) are given as follows (see Fig. 3):

a) if $v > 0$, the $(\mathrm{AcMy})_0$ is converted to $(\mathrm{AcMy})_1$ and the corresponding rate is

$$\omega_{10} = \frac{\mathrm{d}}{\mathrm{d}t}([\mathrm{AcMy}]_1)_{\mathrm{mech}} = \frac{v}{d}[\mathrm{AcMy}]_0. \tag{5}$$

where d is an average distance of displacement of the center of mass of the actin in this transition (see Fig 3).

b) if $v < 0$, the $(\mathrm{AcMy})_1$ is converted back to $(\mathrm{AcMy})_0$ and the corresponding rate is

$$\omega_{01} = \frac{\mathrm{d}}{\mathrm{d}t}([\mathrm{AcMy}]_1)_{\mathrm{mech}} = \frac{v}{d}[\mathrm{AcMy}]_1. \tag{6}$$

Notice that $\omega_{01} < 0$. We can summarize both equations (5) and (6) by defining

$$\begin{aligned} \omega &\equiv \frac{\mathrm{d}}{\mathrm{d}t}([\mathrm{AcMy}]_1)_{\mathrm{mech}} \\ &= \frac{v}{d}\Theta(v)[\mathrm{AcMy}]_0 + \frac{v}{d}\Theta(-v)[\mathrm{AcMy}]_1. \end{aligned} \tag{7}$$

where $\Theta(u)$ is the Heaviside function

$$\Theta(u) = \begin{cases} 0, & \text{if } u < 0, \\ 1, & \text{if } u > 0. \end{cases}$$

We still have to determine the law of motion of the actin filament. The total force on the center of mass is given by

$$F + f_0[\mathrm{AcMy}]_0 + f_1[\mathrm{AcMy}]_1, \tag{8}$$

where F is the total exterior force, which includes the load of the muscle and a spring-like force which tends to bring back elastically the muscle (or the walls of the sarcomer) to their positions in the absence of neural firing and of external load.

We assume a high friction limit for the motion of the center of mass of the actin filament, namely the velocity v of the center of mass (and consequently of the attached myosin heads) is proportional to the total force given by Eq.(8).

The equation of motion is thus

$$\alpha \cdot v = F + f_0[\mathrm{AcMy}]_0 + f_1[\mathrm{AcMy}]_1, \tag{9}$$

where α is a friction coefficient.

The state of the myosin evolves according to the following (non linear) differential equations deduced from Eq. (2 - 4)

$$\frac{\mathrm{d}}{\mathrm{d}t}[\mathrm{My}] = (k_0 + \bar{k}_1)[\mathrm{My}] + \bar{k}_0[\mathrm{AcMy}]_0, \tag{10}$$

$$\frac{\mathrm{d}}{\mathrm{d}t}[\mathrm{AcMy}]_0 = k_0[\mathrm{My}] - \bar{k}_0[\mathrm{AcMy}]_0 - \omega, \tag{11}$$

together with Eq. (9) and the definition of ω in Eq. (7).

Here we have used the obvious result that

$$\frac{\mathrm{d}}{\mathrm{d}t}[\mathrm{AcMy}]_0\Big|_{\mathrm{mech}} = -\frac{\mathrm{d}}{\mathrm{d}t}[\mathrm{AcMy}]_1\Big|_{\mathrm{mech}} = -\omega.$$

Moreover, we can eliminate $[\mathrm{AcMy}]_1$ using the conservation law (1).

$$[\mathrm{AcMy}]_1 = \mathrm{M} - [\mathrm{My}] - [\mathrm{AcMy}]_0. \tag{12}$$

Eq. (11) is non linear, due to the term $-\omega$ and Eq. (7) for ω, when we eliminate v in term of the concentration $[\mathrm{AcMy}]_j$, using the equation of motion Eq. (9). Nevertheless, because ω is 0 if v is 0, the second member of Eq. (11) is a continuous function of the concentration, although its derivatives are discontinuous for $v = 0$.

In the following calculations, we take

$$f_1 = 0. \tag{13}$$

We eliminate v from Eq. (9) with $f_1 = 0$ and we determine $[\mathrm{AcMy}]_1$ using Eq. (12), then

$$\omega = (\alpha \cdot d)^{-1}(F + f_0[\mathrm{AcMy}]_0)\{\Theta(v)[\mathrm{AcMy}]_0 + \Theta(-v)(\mathrm{M} - [\mathrm{My}] - [\mathrm{AcMy}]_0)\}. \tag{14}$$

Define

$$x = [\mathrm{AcMy}]_0; \qquad y = [\mathrm{My}], \tag{15}$$

$$\frac{\mathrm{d}x}{\mathrm{d}t} = -\left(\frac{F}{\alpha d} + \frac{f_0 x}{\alpha d}\right)[\Theta(v)x + \Theta(-v)(M - x - y)] + k_0 y - \bar{k}_0 x \tag{16}$$

$$\frac{\mathrm{d}y}{\mathrm{d}t} = -(k_0 + \bar{k}_1 + k_1)y + (\bar{k}_0 - k_1)x + k_1 M \tag{17}$$

$$\Theta(v) = \Theta\left(\frac{F}{\alpha d} + \frac{f_0}{\alpha d}x\right). \tag{18}$$

We can renormalize these equations using

$$K = k_0 + k_1 + \bar{k}_1 \qquad t^* = Kt$$

$$k_j^* = k_j/K \qquad \bar{k}_j^* = \bar{k}_j/K$$

$$F^* = \frac{F}{\alpha d K} \qquad f_0^* = \frac{f_0}{\alpha d K}$$

so from Eq. (16) and (17), one deduces

$$\frac{\mathrm{d}x}{\mathrm{d}t^*} = -(F^* + f_0^* x)\{\Theta(v)x + \Theta(-v)(M - x - y)\} + k_0^* y - \bar{k}_0^* x \tag{19}$$

$$\frac{\mathrm{d}y}{\mathrm{d}t^*} = \quad - y + (\bar{k}_0^* - k_1^*)x + k_1^* M \tag{20}$$

$$\Theta(v) = \Theta(F^* + f_0^* x)\,. \tag{21}$$

Then,

$$0 \le k_0^* + k_1^* \le 1, \tag{22}$$

and we shall assume that

$$F \le 0, f_0 \ge 0, \tag{23}$$

(see Fig 2. for the sign convention).

STATIONARY POINTS AND STABILITY

In this section, we study the stationary points of the system of Eqs. (19) and (20). The discussion is standard except for the presence of the Heaviside function. We also suppress the * in Eqs. (19) and (20). Whatever is the sign of v, the equation Eq. (17) gives

$$\hat{y} = \bar{k}_0 x + k_1(M - x)\,, \tag{24}$$

for a stationary point ($\hat{x}$, $\hat{y}$).

First, we assume that the corresponding velocity $\hat{v}$ is positive. Then, the stationary solution ($\hat{x}$, $\hat{y}$) satisfies Eq. (24) and the stationary equation corresponding to the equation Eq. (20) for $v > 0$, namely,

$$(F + f_0\hat{x})\hat{x} + k_0 y \quad \bar{k}_0\hat{x} = 0\,. \tag{25}$$

Combining with Eq. (24), one sees that there is only one positive solution for $\hat{x}$. We assume that the velocity $\hat{v}$ is positive or

$$F + f_0\hat{x} > 0\,. \tag{26}$$

A standard analysis shows that *the stationary point* ($\hat{x}$, $\hat{y}$) *is an attractor*.

It can also be proved that, under the natural hypothesis of Eq. (25), if one assumes the condition (26), *there is no stationary solution with a negative velocity*, and that *condition* (26) *is equivalent to the inequality*

$$|\,F\,| \le Mf_0\,. \tag{27}$$

This condition is natural: it means that the external force $-|F|$ (which is negative) has its modulus less than the maximal force Mf_0 which can be exerted by all the myosin heads on the actin filament. This maximal force is exerted when all the myosin heads are attached to the actin filament, in configuration 0, so that the force is Mf_0 in this case.

Finally we have proved that the stationary points satisfies the physical condition

$$\hat{x} + \hat{y} \le M\,, \tag{28}$$

and that, during the evolution, the state of the system stays in the physical region

$$x \ge 0, y \ge 0, x + y \le M,$$

and it is attracted towards the stationary state ($\hat{x}$, $\hat{y}$).

WORK AND EFFICIENCY

The work per unit time, which can be extracted from the system is $w = |F|\,\hat{v}$ in the stationary state, which can be written as

$$w = -(F + f_0\hat{x})\hat{v} + f_0\hat{x}\hat{v}\,.$$

Now the stationary velocity $\hat{v}$ is given by Eq. (9)

$$\hat{v} = \frac{1}{a}(F + f_0\hat{x})\,, \tag{29}$$

so

$$w = f_0\hat{x}\hat{v} - \alpha\hat{v}^2\,. \tag{30}$$

Now the work w_0 per unit time of the force exerted by the myosin heads is

$$w_0 = f_0\hat{v}[\widehat{\mathrm{AcMy}}]_0 = f_0\hat{v}\hat{x}\,. \tag{31}$$

From the first equation (1) for the stationary state, one has

$$(F^* + f_0^*\hat{x})\hat{x} = k_0^*\hat{y}$$

so that restoring normal units and using Eq. (29)

$$\hat{v}\hat{x} = dk_0\hat{y}$$

and thus from Eq.(31)

$$w_0 = f_0 dk_0\hat{y}\,. \tag{32}$$

On the other hand, the consumption of ATP's energy per unit time is the rate of detachment $k_1[\mathrm{AcMy}]_1$ multiplied by the energy of one molecule of ATP, say ε, so the consumption of ATP's energy per unit time is

$$e = k_1[\widehat{\mathrm{AcMy}}]_1\varepsilon.$$

We use the fact that in the stationary state, Eq. (10) gives

$$k_1[\widehat{\mathrm{AcMy}}]_1 = (k_0 + k_1)[\widehat{\mathrm{My}}]\,,$$

so

$$e = (k_0 + k_1)\hat{y}\varepsilon\,. \tag{33}$$

The efficiency is thus the quotient of w by e

$$e = (k_0 + k_1)\hat{y}\varepsilon\,. \tag{33}$$

Notice that in our approximation $f_0{\cdot}d$ is the work of the force f_0 of an individual myosin head when it transits from configuration 0 to configuration 1, and thus it is obviously less than ε (see Fig 3.), so in Eq. (34), the term $(f_0 d/\varepsilon){\cdot}k_0/(k_0 + k_1)$ is less than 1.

The second term of Eq.(34) represents the loss of efficiency due to friction. Thus, Eq. (34) gives the following, simple upper bound for the muscle efficiency:

$$R < \frac{k_0}{k_0 + k_1}\,.$$

DISCUSSION AND CONCLUSION

The model of muscular contraction that we have presented in this article is extremely simple: it is a deterministic kinetics with two degrees of freedom, essentially the number of unattached myosin heads and the number of attached myosin heads before they release to their equilibrium position. Nevertheless, this model respects the obvious physicochemical constraints, reaches a unique stationary state, and captures the main features of the biochemistry of muscular contraction. The parameters in the model are control parameters which depends of other parts of the metabolism of the cell and its environment, namely

(i) the concentration of ATP which is assumed to be given in our model and is the main control parameters for the state of detachment of the myosin heads and provides the energy,

(ii) the level of neural firing, and then the concentration of Ca^{2+} ions which is the main control parameters of the rates of detachment of the myosin heads on the actin filament.

In particular, the level of firing controls the stationary velocity $\hat{v}$ of the actin filament, through the parameter k_0 (rate of attachment) and the total number of sites of the actin which are available for an attachment of a myosin heads, so that this is a control of the number M of "effective" myosin heads.

The number M can be such that $F + f_0 \cdot M$ is very small, so that in this case, although M can be large, the resulting velocity $\hat{v}$ is negligible: this is the case of a concentration with no net work of the force F.

We have neglected many components of the metabolism of the muscular cell. Firstly, we have neglected the global spring force on the muscle, so that our model can represent a muscle starting to contract after being loaded and reaching quickly an asymptotic velocity, which obviously cannot be maintained for ever and is counteracted by the spring force tending to bring back the muscle to its rest conditions. A fully microscopic model was introduced in [11] and solved using a Markovian approximation. However, in order to include specific chemical reactions in the model, all fluctuations effects have been neglected in the present paper.

Another fact is that ATP is the universal energy source of the cell. As a consequence, it is also used to get rid of the waste products of the contraction of the muscle, including lactate, and also to keep the pH in physiological bounds. These two chemical components are at the origin of the muscular fatigue and should be taken into account in more refined calculations.

REFERENCES

[1] Murray, R.; Granner, D.; Mayer, P. and Rockewell, V.: *Précis de Biochimie de Harper*. De Boeck Université, Presses de l'Université de Laval. Traduction de Harper's Biochemistry, 1995. See in particular ch. 58, p.740,

[2] Michal, G.: *Biochemie Atlas*. Spektrum Akad. Verlag, 1991. See in particular ch. 17, p.217,

[3] Hill, T.L.: *Theoretical formalism for the sliding filament model of contraction of striated muscle Part I*. Progress in Biophysics & Molecular Biology **28**, 267-340, 1974,

[4] Hill, T.L. and Eisemberg, E.: *Can free energy transduction be localized at* some *crucial part of the enzymatic cycle*? Quarterly Reviews of Biophysics **14**, 463-511, 1981,

[5] Hill, T.L.: *Some general principles in free energy transduction*. Proceedings of the National Academy of Sciences of the United States of America – Physical Sciences **80**, 2922-2925, 1983,

[6] Hill, T.L.: *Free energy transduction and biochemical cycle kinetics*. Springer, New York, 1981,

[7] Jülicher, F.; Ajdari, A. and Prost, J.: *Modeling molecular motors*. Reviews of Modern Physics **69**, 1269-1281, 1997,

[8] Jülicher, F. and Prost, J.: *Cooperative Molecular Motors*.
Physical Review Letters **75**, 2618-2622, 1995,

[9] Parmeggiani, A.; Jülicher, F.; Ajdari, A. and Prost, J.: *Energy transduction of isothermal ratchets: Generic aspects and specific examples close to and far from equilibrium.*
Physical Review E **60**, 2127-2140, 1999,

[10] Magnasco, M.: *Forced thermal rachets*.
Physical Review Letters **71**, 1477-1481, 1993,

[11] Gaveau, B.; Moreau, M. and Schuman, B.: *Microscopic model of the actin-myosin interaction in muscular contractions*.
Physical Review E **69**, 011108, 2004.

KINETIČKI MODEL KONTRAKCIJE MIŠIĆA

B. Gaveau[1], M. Moreau[2] i B. Schuman[2]

[1]Laboratorij za analizu i matematičku fiziku
Pariz, Francuska

[2]Laboratorij za teorijsku fiziku fluida
Pariz, Francuska

SAŽETAK

U ovom radu izgrađujemo samosuglasni, deterministički model srednjeg polja za opis kontrakcije mišića. Dvije glavne varijable su broj glava miozina i broj glava miozina pričvršćenih za aktin, neposredno nakon pričvršćenja. Model je prirodan u smislu da zadovoljava prirodne fizikalno-kemijske uvjete. Određujemo stacionarno stanje, dokazujemo da je stabilno i određujemo učinkovitost.

KLJUČNE RIJEČI

kontrakcija mišića, kemijska kinetika, učinkovitost